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Daily Stress and Smoking.

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DAILY STRESS AND SMOKING

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ABSTRACT

This study examined daily minor stress and cigarette smoking in adult habitual smokers. In this study, 55 subjects monitored daily stress, daily state anxiety, and daily cigarette intake for 21 consecutive days. Subjects also completed measures of trait anxiety, self-reported smoking motives, recent major life events, and social support. These variables, along with gender, were used to predict associations between daily cigarette intake and scores on daily stress and anxiety inventories. Results of within- and between-subjects time series correlational analyses showed significant associations between scores on measures of daily stress and daily cigarette intake. While subjects as individuals showed marked variability in their associations between daily stress and cigarette consumption, these asssociations could not be predicted by any of the hypothesized predictor variables, including gender, trait anxiety, self-reported negative-affect-reduction smoking, recent life events, and social support. Results of this study are discussed with regard to research and theory in the areas of stress and cigarette smoking. Future research and clinical implications also are discussed.

INTRODUCTION

Overview

Cigarette smoking is considered a major risk factor for some of the leading causes of death in the United States, namely cardiovascular disease, cancer, and lung disease (U.S. Department of Health, Education, and Welfare [USDHEW], 1979; U.S. Department of Health and Human Services [USDHHS], 1984, 1989). In 1979, smoking was deemed the single most preventable cause of illness and death in this country (USDHEW, 1979). Figures from the 1980s suggest that quitting smoking would prevent 25% of all cancer deaths and 350,000 premature deaths from myocardial infarction (American Heart Association, 1988; Fielding, 1985).

Quitting smoking also would result in a decrease in the high costs of health care in the United States. As of 1982 Americans spent over $400 billion per year on health and illness (Matarazzo, 1982), much of which went to the longer-term care required for such chronic, smoking-related illnesses as cancer and heart disease (Taylor, 1990). According to 1980s estimates, in the United States alone, the direct and indirect cost of smoking-related medical care and lost productivity is about $37 billion per year (Warner, 1983).

Despite widespread knowledge of the physical and financial costs of smoking, approximately 54 million Americans were habitual smokers as of 1985 (Fielding, 1985). In addition, an increasingly large proportion of smokers is smoking heavily (Wetterer & von Troschke, 1986). Unfortunately, most smoking cessation programs do not result in long-term behavior change (Marlatt & Gordon, 1985). Reported relapse rates consistently are in the range of 50% to 90% (Marlatt & Gordon, 1980, 1985).

The difficulty in altering smoking behavior has led to a search for intervening psychological factors. While many potential factors have been explored, stress appears particularly important, due to its well-documented links both with smoking (e.g., Borland, 1990; Cummings, Jaen, & Giovino, 1985; O'Connell & Martin, 1987; Shiffman, 1982, 1984, 1986) and illness (Nowack, 1989; Wiebe & McCallum, 1986). Indeed, one major theory of stress proposes that stress leads to illness through its effects on such health-risk behaviors as smoking (e.g., Wiebe & McCallum, 1986).

Most studies of stress and smoking emphasize the effects of laboratory stressors, naturally occuring major life events, or subjective distress on smoking

behavior. In general, these studies have established a role for stress in smoking. However, at present little is known about the relation between daily minor stress and smoking, or potential factors involved in that relation. Though as yet relatively unexplored, daily stress-related changes in smoking might, over time, increase the risk of developing illness or experiencing an exacerbation of a chronic condition. Furthermore, the combined effects of greater stress and increased smoking might intensify health risk.

The introduction of this paper reviews relevant literature on cigarette smoking, particularly its relation to illness and its proposed etiological bases. Stress and its role in smoking then is discussed. A second main section of the paper presents in detail a two-part study which explored relations between daily stress and smoking and attempted to predict potential factors involved in those relations. Results of the study are presented in the third section of the paper. The fourth and final section reviews these results and explores their research and clinical implications.

Cigarette Smoking

Epidemiology

As of 1985, smoking was practiced by almost one fourth of the population of this country (Fielding, 1985). While the proportion of smokers in the U.S. actually has declined since the mid-1960s, when the first Surgeon General's report was released, absolute cigarette consumption has since increased (Wetterer and von Troschke, 1986). This change largely reflects an increase in the proportion of heavy smokers (over 25 cigarettes per day) within the general population of smokers.

Today, as in the past, more men than women smoke cigarettes. However, epidemiological surveys over the past 30 years show that the proportion of women smokers is increasing and is now near that of men (Biener, 1987). Among female smokers, the proportion of heavy smokers also has increased. While American men still outnumber women in developing the key life-threatening chronic illnesses (Verbrugge, 1985), there has been a serious increase in the number of cases of lung cancer among women, and this presumably is due to increased rates of cigarette consumption by women (Biener, 1987).

Another recent trend in cigarette consumption is increasing preference for filter and "low tar" cigarettes. This change in preference has coincided with the noted change in absolute consumption of cigarettes, i.e., to considerably greater consumption of cigarettes per smoker. While the relation may be mere coincidence, it more likely reflects smokers' intensified consumption of cigarettes containing less tar and nicotine, as smokers have been shown to take larger and more frequent puffs when smoking relatively weaker cigarettes (Ashton & Stepney, 1982).

Smoking and Illness

Cigarette smoking clearly is related to illness and death. Cigarette smokers have higher death rates than nonsmokers, regardless of age or sex (USPHS, 1979). Furthermore, mortality risk increases with increasing levels of tobacco consumption and with earlier age at initiation (USDHHS, 1983). A male smoker consuming over two packs per day has a reduced life expectancy of approximately 8 years (Mangan & Golding, 1984).

Smoking is a major risk factor for heart disease. Such large-scale epidemiological studies as the Framingham Heart Disease Epidemiology study and the

National Cooperative Pooling Project have shown a two-to-four-fold increase in risk of coronary heart disease (CHD) mortality among smokers (USDHHS, 1983). Today it is estimated that approximately 30% of the 565,000 annual deaths from CHD are due to smoking (USDHHS, 1989). Cigarette smoking also is the strongest risk factor for sudden cardiac death (Dawber, 1980).

Equally alarming is the annual number of cancer-related deaths attributable to cigarette smoking. According to 1980s figures, in the United States, approximately 412,000 persons die of cancer each year; of these, about 125,000 are smoking-related (USDHHS, 1983). In fact, smoking is the leading cause of cancer mortality in this country, with smokers carrying twice the risk of nonsmokers. Almost 90% of lung cancer deaths are caused by smoking (USDHHS, 1989). Smoking also greatly increases risk of cancers of the larynx, esophagus, oral cavity, and bladder (McCoy, Hecht, & Wynder, 1980; Wigle, Mao, & Grace, 1980). The risk of oral cancer is compounded for smokers who drink heavily (McCoy & Wynder, 1979).

Lung disease is another major disease category linked to cigarette smoking. Smoking is the leading cause of chronic obstructive lung disease (COLD), with

smokers comprising close to 90% of all COLD deaths (USDHHS, 1984). As noted in Brantley and Garrett (1991), each year there occur over 19,000 smoking-related deaths from pulmonary disease, including emphysema, bronchitis, and COLD.

Finally, cigarette smoking is known to interact in a multiplicative fashion with other risk factors for illness, particularly heart disease (Johnston, 1989). For example, smokers with high blood pressure or elevated serum cholesterol have about three times the risk of cardiovascular disease as do smokers without the other risk factors (Kannel, 1976). Also, the risk of mortality from myocardial infarction (MI) is substantially increased in smokers who are obese (Heyden, Cassel, Bartel, Tyroler, Hames, & Cornoni, 1971) and in women who both smoke and use oral contraceptives (Pettiti, Wingerd, Pellegrin, & Ramcharan, 1979).

Psychology of Smoking

Biological Factors

Relevant biological factors in smoking include the psychopharmacology and chemical actions of nicotine, and the paradoxical biphasic effects of nicotine on the autonomic and central nervous systems. These factors,

briefly discussed below, are incorporated in biological theories of smoking.

Psychopharmacology of nicotine Of the hundreds of chemicals identified in cigarette smoke, nicotine appears to be the most important pharmacological agent. As discussed by Ashton and Stepney (1982), smokers rarely smoke cigarette-like substances that do not contain nicotine, and tend not to smoke tobacco cigarettes containing a very low amount of nicotine. This suggests that nicotine has powerful reinforcing properties that may, in part, underlie smoking behavior.

When cigarette smoke is inhaled, nicotine is rapidly and efficiently absorbed, producing almost immediate but short-lived effects. Notably, the nicotine from one cigarette puff reaches the brain in about 7 seconds. The adrenal medulla and the sympathetic ganglia, both important parts of the autonomic nervous system, also take up nicotine quite readily. The rapid uptake of nicotine by these nervous tissues helps explain the immediacy with which psychological effects are achieved.

Inhaling cigarette smoke causes nicotine levels to rise very quickly in the bloodstream. Blood nicotine

levels peak around the time a cigarette is extinguished and decline until the next cigarette is consumed. However, the frequent and regular consumption of cigarettes has a cumulative effect, so that blood levels of nicotine remain higher than baseline even during troughs. In this way a smoker can maintain a high blood nicotine level by continuing to smoke additional cigarettes. Also, there is evidence that each puff causes its own slight peak in blood nicotine concentration (Ashton & Stepney, 1982).

Nicotine is rapidly metabolized and excreted from the body. The pH balance of the urine appears to play some role in the rate of excretion. As nicotine is an alkaloid, when urinary pH is high (alkalized), the rate of excretion is slower; when it is low (i.e., acidic), nicotine is excreted more rapidly, causing blood nicotine levels to fall. Acidifying the urine of smokers (i.e., by administering bicarbonate of soda or exposing them to a stressor) has been shown to increase smoking frequency, presumably due to enhanced excretion of nicotine (Schachter, Kozlowski, & Silverstein, 1977a; Schachter, Silverstein, Kozlowski, Herman, & Liebling, 1977b; Schachter, Silverstein, & Perlick, 1977c).

Chemical actions of nicotine Nicotine is structurally similar to acetylcholine (ACh), and is thereby accepted by certain ACh receptors (nicotinic receptors), where it can exert ACh-like actions. Further, by combining with ACh receptors, nicotine can block the receptors from receiving nerve impulses (Ashton & Stepney, 1982). This synaptic process helps explain the biphasic (i.e., both stimulant and depressant) effects of nicotine's actions on the body.

When nicotine first combines with a nicotinic receptor the effect is, like that of ACh stimulation, excitatory. However, with large doses of nicotine the effect of nicotine at ACh-synapses is depressing, due to the blockage of ACh transmission by the nicotine molecule. In this way nicotine can produce rapid, reversible, biphasic effects. However, the nature of the synaptic effects of nicotine (i.e., inhibitory, excitatory, or both) depends on the dose administered, and is affected by such puff dimensions as size, duration, and depth (Ashton & Stepney, 1982).

Paradoxical effects of nicotine on the nervous system The biphasic chemical and bodily effects of nicotine are paradoxical, particularly in light of a proposed stress-smoking relation. If smoking produces

bodily and cortical arousal, as evidence suggests, it should not reduce stress or arousal, but rather should enhance them. However, nicotine commonly is found to decrease both self-reported and behavioral indices of emotional arousal, with some smokers reporting that nicotine makes them feel more tranquil (Gilbert, 1979). This issue has been termed in the literature "Nesbitt's paradox" (Schachter, 1973). The paradox is further complicated by smokers' varied reports of their reasons for smoking. While many smokers claim to smoke primarily to achieve emotion reduction or pleasurable relaxation, a proportion of smokers report smoking for stimulation (Ikard, Green, & Horn, 1969; Ikard & Tomkins, 1973).

Attempts to resolve these paradoxes have established some consistent findings. First, as discussed by Gilbert (1979), it is clear from both human and animal studies that nicotine does produce widespread autonomic arousal. In humans, even mild cigarette consumption (one or two cigarettes) causes significant sympathomimetic symptoms, most notably increases in resting heart rate, blood pressure, serum levels of epinephrine and adrenocortical compounds, and vasoconstriction. This appears true whether subjects

are in quiescent or moderately aroused states prior to smoking. However, at the same time, nicotine also produces relaxation of the reflexive muscles. Decreased muscular tension thus may be experienced by the smoker as tranquilization, despite other signs of autonomic arousal.

Dual effects of nicotine also are seen on measures of cortical arousal. In both humans and animals, smoking-sized doses of nicotine produce short-term CNS arousal, as indicated by increased EEG activity. However, more sophisticated measures reveal mixed arousing and depressing effects of nicotine, and sometimes show overall cortical sedation (Gilbert, 1979). At the same time, nicotine deprivation is associated with EEG sedation or depression, but, paradoxically, also is accompanied by feelings of restlessness and dysphoria (Gilbert, 1979).

Several biological theories have been offered to explain the paradoxical cortical effects of nicotine. For example, Eysenck (1973) suggests that such personality factors as extraversion and/or the smoker's preexisting level of cortical arousal determine whether nicotine's effects are perceived as arousing or sedating. Miller (1973) proposed that nicotine acts to

inhibit aggression, fear and other emotions by inhibiting muscarinic receptors. A third model, the glucocorticoid-ACTH model, suggests that release of glucocorticoids induced by nicotine mediates or causes a reduction of emotional behavior (e.g., Andersson, 1975; Hill & Wynder, 1974). While each of these theories has some support, the precise mechanisms by which nicotine produces its paradoxical effects remain unknown (Gilbert, 1979).

Psychological Models of Smoking

Biological models of smoking address the psychopharmacology and biochemistry of nicotine, but typically do not explore behavioral and emotional factors involved in smoking behavior. To that end, several psychological and biobehavioral models have been offered to explain why people smoke. For example, psychoanalytic views emphasize the role of cigarettes in oral erotic gratification and displacement activity to cope with inner conflict (Ashton & Stepney, 1982; Krantz, Grunberg, & Baum, 1985). Such views are largely theoretical, however and have little empirical basis. Other models also have been offered, and are based primarily on biological, behavioral and psychosocial factors in smoking. Though by no means

mutually exclusive, these models can be grouped as follows: (1) nicotine-regulation, (2) psychological tool, (3) social learning, and (4) affect-reduction.

Nicotine-regulation models Nicotine-regulation models of smoking state that habitual smokers smoke primarily in order to maintain a steady level of nicotine in the bloodstream (Feuerstein, Labbe, & Kuczmierczyk, 1986). This may reflect physiological dependence (Jarvik, 1979) and/or an attempt to avoid the aversiveness of withdrawal (Russell, 1979; Schachter et al., 1977c; Schachter, 1978).

There is ample support for a model of smoking based on self-regulation of nicotine. For example, studies with humans have shown that smokers will increase their smoking intensity when given cigarettes with lower nicotine content (Schachter, 1977, 1978; Stepney, 1980) or when their urine has been acidified (Schachter et al., 1977a,b). In some studies, smokers also have been shown to smoke less when given nicotine intravenously or in alternate form such as chewing gum (e.g., Lucchesi, Schuster, & Emley, 1967). In addition, laboratory animals have been shown to self-administer nicotine, presumably in an attempt to regulate blood levels (Ashton & Stepney, 1982).

The demonstration that both humans and animals will self-regulate their blood nicotine levels supports a nicotine-regulation model of habitual smoking. However, psychosocial and contextual factors also appear important. This was demonstrated by Schachter and colleagues (Schachter et al., 1977c), who found that both light and heavy smokers smoked more in certain situations, including at parties and during intensive, presumably stressful academic exercises and presentations. In addition, it is clear that smokers far prefer cigarette inhalation to other forms of nicotine administration, and do not always smoke less when given alternate forms of nicotine (e.g., Jarvik, Glick, & Nakamura, 1970; Kumar, Cooke, Lader, & Russell, 1977; Turner, Sillett, Taylor, & McNicol, 1977).

Because factors other than the pharmacology or addictiveness of nicotine appear important in smoking behavior, a pure nicotine-regulation or addiction model may be too simplistic. Accordingly, the model has been expanded by Leventhal and Cleary (1980) to account for the role of nicotine in regulating emotional state. These authors' multiple regulation model suggests that departures from a homeostatic hedonic or emotional

state, rather than blood or plasma nicotine levels per se, stimulate smoking behavior. In this model such factors as craving or emotional distress may induce smoking, though the link between these internal cues and drops in nicotine level has yet to be delineated.

Leventhal and Cleary (1980) further propose that smoking serves as a coping skill to minimize unpleasant emotions and enhance relaxation or pleasure. They also suggest that an "emotional memory" for events previously associated with smoking may serve as cues to smoke in later, similar situations (e.g., under stress).

Psychological tool model The "psychological tool model" of smoking (Ashton & Stepney, 1982), is based on such desirable short-term psychopharmacologic effects of nicotine as increased attention, relaxation, and emotion-reduction. This model posits that, independent of one's initial reasons for smoking, smoking behavior becomes habitual through repeated attempts to gain cognitive rewards and to manipulate psychological state under various environmental conditions.

Experimental studies of the effects of nicotine deprivation on smokers' behavior provide some support for the psychological tool model. For example, Gilbert

(1979) reviews evidence that smokers deprived of cigarettes demonstrate improved cognitive task performance and enhanced mood state when allowed to smoke. Smokers allowed to smoke or given higher nicotine content cigarettes were also found by Nesbitt (1973) to better tolerate electric shocks than were smokers forbidden to smoke or given low nicotine cigarettes. These results suggest that direct, desirable pharmacologic effects of nicotine enhance both performance and mood under stressful conditions, and in this way reinforce continued smoking.

Social learning model Social learning theory views smoking in the context of social reinforcement and conditioning (Feuerstein et al., 1986). From this perspective, the immediate social rewards of initial smoking (e.g., peer acceptance) outweigh possible long-term adverse consequences. Socially desirable images associated with smoking (e.g., strength, power, sophistication) also may reinforce initial smoking behavior (Krantz et al., 1985). However, with repeated smoking, the consumption of cigarettes is thought to become associated with various stimuli ranging from social interaction cues to other chemicals to feelings of anxiety or distress (Ashton & Stepney, 1982).

According to this model, smoking behavior then may be maintained by its learned associations with multiple internal and external cues, including emotional states and stress (Feuerstein et al., 1986).

In support of a social learning model of smoking, a number of studies have reported that stress or negative emotional states commonly precipitate relapse (e.g., Marlatt, 1985; Marlatt & Gordon, 1980; Ockene, Benfari, Nuttall, Hurwitz, & Ockene, 1982; Ockene, Nuttall, Benfari, Hurwitz, & Ockene, 1981; O'Connell & Martin, 1987; Shiffman, 1982, 1984; Tunstall, Ginsberg, & Hall, 1985). Social situations, too, have been found significant in triggering relapse in smoking (Marlatt, 1985; Marlatt & Gordon, 1980; Shiffman, 1984, 1986). Situations associated with such familiar food cues as after meals or while drinking coffee or alcohol also are commonly said to precipitate relapse (e.g., Zimmerman, Warheit, Ulbrich, & Auth, 1990). These findings suggest that smoking may be associated with specific environmental, emotional, or physiological cues that continue to elicit smoking urges even when smoking has ceased. The persistence of such cues appears to encourage relapse. Accordingly, many behavioral treatment programs now incorporate

techniques designed to loosen the associations between smoking-cues and smoking (Feuerstein et al., 1986).

Affect-reduction model A fourth psychological model of smoking emphasizes the value of cigarettes in reducing "negative affect," or feelings of emotional distress. This model derives largely from the early work of Tomkins and colleagues (Tomkins, 1966, 1968; Ikard & Tomkins, 1973), who discussed various emotion-regulation functions of smoking in determining smoking "types." Tomkins believed these types reflected different mechanisms that sustain smoking behavior. For some ("positive affect smokers"), smoking was thought to produce positive mood states, while for others ("negative affect smokers"), smoking was thought to neutralize negative mood states. A third type, the addicted (or preaddicted) smoker, was thought to smoke to reduce negative affect associated with deprivation (i.e., craving). The last type discussed is the habitual smoker, for whom smoking no longer serves to regulate affect but has become automatic, perhaps as an outgrowth of addiction.

There is some support for Tomkins's typology. For example, factor analyses on data from several different samples of smokers have effectively differentiated

smoking types corresponding to the four types proposed by Tomkins (Feuerstein et al., 1986; Leventhal & Cleary, 1980). Further support is found in a group of validity studies conducted by Ikard and Tomkins (1973), in which smokers smoked the greatest number of cigarettes under those conditions most consistent with their "type" as measured by a Tomkins-based smoking questionnaire. For example, "negative affect smokers" smoked more during an upsetting film but not during a funny film, and addicted smokers (high scorers on a Psychological Addiction scale) smoked about the same rate during both films. However, while the findings of Ikard and Tomkins are suggestive of real differences in smoking behaviors of various smoking "types," more recent studies examining smoking motives in a naturalistic setting (e.g., Joffe, Lowe, & Fisher, 1981; Shiffman & Prange, 1988; Tate & Stanton, 1990) have not consistently found these same smoking motives to predict actual smoking behavior.

Stress and Smoking

The various biological, biobehavioral and learning models of smoking behavior provide some theoretical basis for a relation between stress and/or negative affect and smoking. Studies directly testing this

relation both inside and outside the laboratory provide further evidence. A review of that literature will be presented, following a general discussion of stress.

Definition of Stress

Stress has been defined as "a state of imbalance within a person, elicited by an actual or perceived disparity between environmental demands and the person's capacity to cope with these demands" (Maes, Vingerhoets, & Van Heck, 1987, p. 567). Stressors, by definition, are demands which elicit a response in the organism. This complex response has physiological, cognitive, and behavioral components.

Models of Stress

Three major models of stress are discussed in the literature. Each focuses on a different component of the stress experience: the response, the stressor, or the interaction between the two.

Response-based models emphasize the organism's reaction to demand or threat. Many of these models are based in the pioneering work of Hans Selye (1956, 1976), who described various physiologic changes that occurred in response to noxious stimuli. Selye's research with laboratory animals showed these effects to be nonspecific, i.e., they occurred regardless of

the type of aversive stimulus administered. He termed this nonspecific reaction to various physical stressors the General Adaptation Syndrome, or GAS, a three-stage reaction comprised of alarm, resistance and exhaustion.

In Selye's model, the effects of stress are seen as cumulative; i.e., damage secondary to stress may accumulate over time. Also, these nonspecific effects may produce serious pathology when the organism's coping resources are overwhelmed. Third, stressors may be additive in nature, i.e., the reaction to previous threat(s) may intensify the response to later stressors (Fleming, Baum, & Singer, 1984).

Despite its widespread influence, Selye's response-based model of stress has been challenged in the literature. As discussed by Sutherland and Cooper (1990), a nonspecific conceptualization of stress may be overly simplistic. More recent evidence suggests there are different patterns of responses to various stimuli, and these responses tend to be stimulus-specific (e.g., Lacey, 1967, and Mason, 1971, cited in Sutherland & Cooper; Mason, 1974). Furthermore, Selye's approach fails to adequately address psychological responses to stress. Also, his approach does not address the possibility that the response to a

potential threat may become the stimulus for a different response (Christian & Lolas, 1985). Other models of stress, however, do take these issues into account.

Stimulus-based models focus on characteristics of environmental events, or stressors, leading to a reaction. These models define stressors as events that place demands on an organism and alter its biological or psychological integrity. Attempts to describe life change events and quantify their stressfulness typify current stimulus-based models (e.g., Holmes & Rahe, 1967; Kanner, Coyne, Schaefer, & Lazarus, 1981). These models examine such dimensions of stressors as magnitude, frequency, intensity, and duration. They also examine qualitative aspects of stressors. For example, evidence suggests that events perceived as undesirable (e.g., Sarason, Johnson, & Siegel, 1978; Vinokur & Selzer, 1975), unpredictable (Matheny & Cupp, 1983), and uncontrollable (Suls & Mullen, 1981) are reported as more stressful. Events perceived as both uncontrollable and undesirable may be most strongly related to physical and psychological disorder (Husaini & Neff, 1978; McFarlane, Norman, Streiner, Roy, & Scott, 1980; Suls & Mullen, 1981).

Interactional models view stress as a fluid transaction between organism and environment (e.g., Folkman & Lazarus, 1980; Folkman, Lazarus, Dunkel-Schetter, DeLongis, & Gruen, 1986). These models account for responses to stressors as well as stressors per se. In such models, stress consists of an event or situation and an organism's appraisal of that event as threatening. That appraisal leads to a response, which in turn modifies the initiating event, and so on.

Measurement of Stressors

The bulk of psychological stress research involves measurement of life events, conceived as measurable external or internal occurrences requiring change or adaptation. Both major and minor events have been studied. A greater frequency or intensity of life events is thought to demand greater adjustment, thereby increasing an organism's vulnerability to illness (Pearlin, Lieberman, Menaghan, & Mullen, 1981). However, the biological mechanisms by which these events produce their effects remain unclear.

Major events The Social Readjustment Rating Scale (SRRS) (Holmes & Rahe, 1967) represents the first attempt to quantify the stressfulness of various major life events. This scale assesses the degree of life

change experienced by an individual over a period of time. Items range from death of a spouse to major personal achievement to minor violations of the law, and are weighted to reflect their relative stress impact. Higher scores reflect greater life change requiring greater adjustment in normal life.

A number of studies have reported a positive relation between higher scores on the SRRS and illness episodes. For example, in an early study by Rahe (1974), navy personnel were asked to report life changes and illnesses over the previous decade. In this retrospective study, subjects reporting fewer life change events in a given year were found to report less illness in the following year. Of those reporting moderate stress, about 50% reported illness in the following year, and among subjects reporting high stress, approximately 70% later experienced illness. Other early studies, both retrospective and prospective, also found significant relations between a greater magnitude of life change, as measured by the SRRS, and greater risk of illness and/or increased likelihood of exacerbation of chronic illness (e.g., Holmes & Masuda, 1974; Rahe & Arthur, 1978; Wyler, Masuda, & Holmes, 1971).

More recent studies generally have confirmed an association between major events and illness (Schroeder & Costa, 1984). In recent years, major life events have been shown to influence the course or development of several serious medical illnesses, including rheumatoid arthritis (Baker & Brewerton, 1981), cancer (Cooper, Davies-Cooper, & Faragher, 1986; Horne & Picard, 1980), heart disease (Byrne, 1987), and diabetes (e.g., Evans, 1985; Goetsch, 1989; Surwit & Feinglos, 1984; Surwit, Feinglos, & Scovern, 1983). A similar association is found between major stress and less severe forms of physical illness. Graham, Douglas, and Ryan (1986), for example, found a positive relation between stress and acute respiratory infection; in that six-month prospective study, subjects with higher scores on major and minor life events scales had more episodes and symptom days than had subjects with lower stress scores. Similar findings have been obtained for exacerbations of peptic ulcer disease (Gilligan, Fung, Piper, & Tennant, 1987), skin disease (Gil, Keefe, Sampson, McCaskill, Rodin, & Crisson, 1987) and various other minor illnesses (Sarason, Levine, & Sarason, 1982).

While the bulk of the major life events research shows a consistent but moderate association between greater life stress and illness onset or exacerbation, it must also be noted that major life events do not account for much of the variance in illness. As noted by Rabkin and Struening (1976), the average correlation between major events and later illness is only around .12. Also, the exclusion of "contaminated" (i.e., outcome-related) items from life events measures tends to decrease the association between events and illness (Schroeder & Costa, 1984). Accordingly, some researchers have turned their attention to minor life events.

Minor events Minor events are conceived as aggravations or irritations of daily life, such as failing to meet a deadline or getting stuck in traffic (Brantley, Waggoner, Jones, & Rappaport, 1987). As these events occur more frequently than do major events, they are thought to have less individual adverse impact (Brantley et al., 1987).

In the past decade or so, researchers have begun reporting a link between minor stress and symptom exacerbations across several illness categories. For example, studies by Brantley and colleagues have shown

significant associations between higher daily stress scores and physical symptoms associated with various chronic illnesses (e.g., Brantley, Everett, Jones, & Sletten, 1990; Goreczny, Brantley, Buss, & Waters, 1988; Nathan, Brantley, Goreczny, & Jones, 1988). These studies typically have used repeated measures designs to assess differences in symptom reports on high versus low stress days. Taken together, the findings support a relation between daily minor stress and illness exacerbations. However, the role of minor stressors in illness remains unclear. While some believe minor events may be the mechanism through which major events produce their impact (e.g., Hinkle, 1974), the correlation between major and minor event scores typically is only modest, ranging from -.27 (Zarski, 1984) to +.49 (Eckenrode, 1984). Also, a number of studies have found minor events to predict somatic and psychological symptoms independent of major life events (e.g., DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; Eckenrode, 1984; Monroe, 1983). These findings suggest that minor events contribute independently to illness (Kanner et al., 1981).

The earliest inventory of minor life events is the Hassles scale (Kanner et al., 1981), a 117-item

inventory that measures the frequency and perceived impact of minor stressors. Designed for use as a periodic measure, the scale asks respondents to recall a number of minor stressful events occurring over the past month. Scores on the Hassles scale have been found to predict both current and later psychological symptoms (DeLongis et al., 1982; Kanner et al., 1981) and overall health status (Zarski, 1984). However, the scale relies on retrospective reports of stressors, which may be clouded by forgetting or bias. Also, the scale does not lend itself to more frequent use, as in tracking daily or weekly fluctuations in events or related symptoms.

A newer minor events measure, the Daily Stress Inventory (DSI) (Brantley & Jones, 1989) overcomes some of these measurement issues. The immediacy with which minor stressors are examined makes the DSI particularly useful in the measurement of daily stressful events over time. The instrument also provides for events that occurred but were not perceived as stressful.

The DSI assesses the frequency and impact of 58 minor stressors occurring over the course of 24 hours. Respondents rate the impact of each item that occurred, using a 7-point Likert-type scale ranging from 1

("occurred but did not cause stress") to 7 ("caused me to panic"). Daily stress frequency, impact and relative impact scores are yielded from these responses.

The DSI appears useful in the assessment of stress-related physical symptoms (e.g., Brantley et al., 1990; Goreczny et al., 1988; Nathan et al., 1988). Higher DSI scores also have been shown to correlate positively with biochemical indices of stress (Brantley, Dietz, McNight, Jones, & Tulley, 1988).

Social Support and Stress

Social support has increasingly been recognized as an important environmental modulator of stress. However, at present it still is unclear what is meant by the term social support. A number of definitions have been offered. For example, Krantz et al. (1985) describe social support in terms of benefits gained from relationships with others. These relationships are thought to provide a sense of belongingness, instrumental aid, emotional comfort, opportunities for social comparison, and enhanced self-esteem. Broadhead and colleagues (Broadhead et al., 1983) define social support simply as resources provided by other persons.

The direct beneficial nature of social support is suggested from several prospective studies with different follow-up periods ranging from 30 months to 12 years; in these studies, mortality rates are found to be higher for those with lower as compared to higher social support (Berkman & Syme, 1979; Blazer, 1982; House, Robbins, & Metzner, 1982; Schoenbach, Kaplan, Fredman, & Kleinbaum, 1986). Studies examining support under stress or crisis situations also clearly show that social support helps to modify the effects of undesirable life events (Bruhn & Phillips, 1984; Cobb, 1976; Cohen & Syme, 1985; Cronkite & Moos, 1984; Hall, Williams, & Greenberg, 1985; Sarason, Sarason, Potter, & Antoni, 1985; Schaefer, Coyne, & Lazarus, 1981; Thoits, 1982; Vaux, 1988). Notably, results have been similar across studies using diverse measures and outcome criteria (Blake, 1988).

Given the consistent relation between higher social support and better outcome in the face of life stressors, many authors have suggested that social support mediates between stress and illness (e.g., Cobb, 1976; Schaefer et al., 1981). However, it is unclear how social support might function in this relation. Social supportive resources may have a

direct positive effect on health, and/or may serve as a buffer against ill effects of stress. Alternately, the superior outcome observed for those with higher social support may simply reflect the comparatively adverse effects of not having support during difficult times (Krantz et al., 1985). At the very least, greater social support may give individuals time to adapt to stress, perhaps by mobilizing coping behaviors and defenses (Bruhn & Phillips, 1984).

As yet no one aspect or component of social support has emerged as the crucial variable in health outcomes (Vaux, 1988). However, emotional support may be of particular value in coping with the stress of smoking cessation (Hanson, Isacsson, Janzon, & Lindell, 1990). Also, having a higher degree of social support from spouses and/or coworkers appears to increase the chance of long-term abstinence after quitting (e.g., Horwitz, Hindi-Alexander, & Wagner, 1985; Mermelstein, Lichtenstein, & McIntyre, 1983; Westman, Eden, & Shirom, 1985).

Proposed Mechanisms in Stress and Illness

Most theories of stress presume that stress weakens resistance to illness, and thereby predisposes to health problems. However, the pathways by which

this occurs remain unknown. One possibility is that stress produces direct psychophysiological effects that influence physical health (Krantz et al., 1985). In other words, psychosocial stimuli may lead directly to changes in tissue function through bodily responses to stressful stimuli. This hypothesis is consistent with Selye's (1976) model of stress as a nonspecific bodily response to external demands, and is supported by studies reporting increased risk of heart disease in persons with characteristics of the Type A Behavior Pattern (Haynes, Feinlieb, & Kannel, 1980; Matthews, 1988; Rosenman et al., 1975). Support for a direct psychophysiological model of stress also is found in studies showing increased morbidity and lower immune functioning in the newly bereaved (e.g., Bartrop, Luckhurst, & Bjorntorp, 1977), in adults faced with threatening life events (Fleming et al., 1984; Irwin, Patterson, Smith, & Caldwell, 1990; Willis, Thomas, Garry, & Goodwin, 1986), and in animals exposed to laboratory stressors (Monjan, 1981). Also, among healthy human subjects, chronic life change has been associated with one or more depressed immune functions (Palmblad, 1981).

While there is support for a direct physiological model of stress and illness, indirect mechanisms also have been proposed. For example, it has been suggested that stress adversely affects health through changes in health-related behaviors (Hinkle, 1974; Jemmott & Locke, 1984; Maes et al., 1987; Nowack, 1989; Sutherland & Cooper, 1990; Wiebe & McCallum, 1986). This hypothesis predicts that higher levels of psychosocial stress are associated with increases in various behaviors associated with health risk. Such behavioral changes may evolve into lifestyles or habits which, over time, increase the likelihood of developing illness (Maes et al., 1987). Also, behavioral changes in response to stress may interact with more direct effects of stress to produce or exacerbate illness (e.g., Dembroski, 1986; Johnston, 1989).

A behavioral risk model of stress and illness is of particular note because many risk factors for illness include or are related to potentially modifiable lifestyle factors. Such behaviors as cigarette smoking, excessive alcohol consumption, poor dietary habits, and sedentary lifestyle all have been linked to major illness (Hamburg, Elliott, & Parron,

1982; Kannel, 1979), as well as to nonbehavioral risk factors for illness (Johnston, 1989).

Of the behavioral risk factors, smoking is most salient in its relation to illness and to stress. As noted, smoking is considered the primary preventable cause of mortality in this country (USPHS, 1976, 1979). Also, there is ample evidence that stress affects multiple aspects of smoking behavior (Abrams, Monti, Pinto, Elder, Brown, & Jacobus, 1987; Aneshensel & Huba, 1983), and so may further intensify associated health risks (Horowitz et al., 1979).

Studies on Stress and Smoking

The classic laboratory studies on stress and smoking were conducted by Stanley Schachter and colleagues in 1977. One experiment in this series showed that subjects exposed to a high-stress condition (painful electric shocks) smoked more cigarettes and took more puffs per cigarette than did subjects in a low-stress condition (Schachter et al., 1977b). Also, the high-stress subjects in this experiment were found to have more acidic urine than did the low-stress subjects, suggesting that greater stress produced faster metabolism and elimination of nicotine (leading to more rapid craving for cigarettes). Interestingly,

in a related experiment, high-stress subjects whose urine was alkalized prior to stress exposure did not smoke more than low-stress subjects (Schachter et al., 1977b).

In another laboratory study of stress and smoking (Mangan & Golding, 1978), smoking was found to modulate arousal level under stressful (loud noise) and nonstressful (relaxation/sensory isolation) conditions. In the stress condition, smoking was associated with increased percentage of alpha activity on the EEG, suggesting reduced arousal. However, in the relaxation condition, actual smoking had the effect of reducing alpha activity, suggesting increased arousal. Sham smoking produced only mild and short-lived effects on arousal, indicating that nicotine itself (and not smoking-related behaviors) caused the change in EEG arousal level. These results illustrate the paradoxical effects of nicotine, and suggest that nicotine ingestion may modulate emotional arousal under stress.

A number of animal studies provide indirect support for the idea that nicotine counteracts the response to stress. For example, Hutchinson and Emley (1973) found that nicotine administration blocked the

suppression of food-acquisition in rats and monkeys conditioned to a tone signaling electric shock. Nelson (1978) found that rats repeatedly injected with nicotine showed significantly less "freezing" when exposed to a cat, as compared to control animals not given the injections. Furthermore, this effect was strongest in the most "emotional" animals.

Thus, the findings of several laboratory studies with humans and animals suggest that smoking increases under stress, that nicotine may serve to normalize arousal level under varying environmental conditions, and that nicotine may counteract a stress response. However, it should be noted that the laboratory itself may be an abnormal, stressful environment which influences the smoking behavior of subjects. For instance, studies by Ashton, Stepney, and Thompson (1978, 1979) found that subjects tended to take greater nicotine doses inside the laboratory compared with outside, as measured by analysis of nicotine retained in cigarette filters.

On the other hand, nonlaboratory studies with humans also have found stress-related changes in smoking behavior. Several studies have reported increased substance use, including cigarette

consumption, in relation to major life stressors in the natural environment. For example, Wills (1986) found a positive relation between stress (major, minor, and acute) and substance use (tobacco, alcohol), in young adolescents. Zisook, Schucter, and Mulvihill (1990) found that loss of a spouse was related to increased cigarette and alcohol consumption and use of psychotropic medication in bereaved men and women. Westman et al. (1985) found that various indices of job stress were positively related to smoking intensity and negatively related to cessation in a large sample of kibbuzim members. Such findings suggest that major life stress may be associated with increased substance consumption, including but not limited to cigarette smoking.

Studies of situations in which smokers are tempted to smoke also cite stress as a factor. For example, Frith (1971) and O'Connor (1980) analyzed situations in which smokers reported temptations to smoke. Both investigators found that while very heavy smokers reported smoking in all sorts of situations, a proportion of smokers reported smoking more under stress. Further, women in particular reported greater stress-related smoking.

There also is good reason to believe that stress is a factor in smoking cessation and relapse. As noted by Hanson et al. (1990), both major and minor stressful situations in daily life appear to increase the risk of relapse or failure in quitting. For example, Gunn (1983) found that continued smoking and dropping out of a cessation clinic was strongly predicted by higher life-stress scores. Caplan, Cobb, and French (1975) found that smokers reported significantly more job stress than did quitters. A commonly reported trigger of relapse is interpersonal conflict (e.g., O'Connell & Martin, 1987), which may be viewed as a form of minor stress. Also, relapse often is reportedly precipitated or accompanied by a negative affective state (e.g., Brandon, Tiffany, Obremsky, & Baker, 1990).

The consistent findings on stress, cessation and relapse imply that individuals who smoke primarily to reduce emotional distress may find it particularly difficult to quit. For instance, smokers who report increased smoking under stress also appear less likely to attempt to quit (Zimmerman et al., 1990). In addition, full relapse (i.e., a return to habitual smoking) has been found more likely when post-cessation smoking urges are accompanied by negative

affect, particularly anxiety (Brandon et al., 1990; O'Connell & Martin, 1987).

Finally, stress and smoking may interact to further increase risk of illness. This possibility was explored in a set of well-designed experiments by Dembroski, MacDougall, and colleagues (Dembroski, 1986), who examined the combined effects of laboratory stress and smoking on cardiovascular response. Using a 2 x 2 factorial design, they found that subjects who smoked and then engaged in a stressful game showed twice the magnitude of blood pressure and heart rate increases as did subjects who only smoked or only played the stressful game. Similar results later were obtained with female subjects. Also, in both cases the effect appeared synergistic rather than additive. Based on these and related findings, Dembroski (1986) suggests that stress-related smoking may increase risk for a CHD event, particularly for those who are hyperreactive to stress or to smoking.

Daily stress and smoking While there is a large literature on stress and smoking, few studies have explored the role of daily minor stress. To date, only one study has specifically examined the effects of changing daily stress level on cigarette smoking in a

real-life setting (Conway, Vickers, Ward, & Rahe, 1981). This longitudinal field study investigated the impact of occupational stress on self-reported cigarette, coffee and alcohol consumption, using a sample of 34 U.S. Navy petty officers assigned to a Naval Training Center in San Diego. The study was conducted over a period of 8 months, during which time there were known systematic variations in stress level associated with training and assignment to a recruit company. The 14 study days used for data analysis comprised the first and last days of Company Commander School and 6 days during each of two recruit-training cycles, reflecting two different levels of occupational stress.

In this repeated measures design, high-stress days were associated with significantly higher cigarette consumption than were lower-stress days. Furthermore, the relation between daily stress and smoking was stronger than that between stress and caffeine or alcohol consumption. Finally, results showed that habitual cigarette smoking and coffee drinking were positively associated with chronic tendencies to perceive high stress.

A major strength of the Conway study was the natural "manipulation" of stress level. That there were known differences in stressfulness of study days, confirmed by the subjects' self-report of stress differences, suggests that real differences in work-stress level were in fact associated with significant changes in cigarette consumption in the natural environment. However, the Conway study was limited in that the sample was restricted to males and to navy officers; generalizability of findings to female subjects and to civilians, particularly in a naturalistic setting, is difficult to assess. In addition, the investigators did not assess more typical minor stressors as might be encountered by a given smoker on a given day. Another weakness was use of retrospective estimates of daily cigarette consumption over the preceding week as a major dependent variable.

Despite these limitations, results of the Conway study suggest that greater daily stress is associated with increases in daily smoking rates in a proportion of subjects. However, as with other findings on stress and smoking, factors underlying the association are unclear, and individual differences between subjects may have played a role.

Gender and affect in smoking and stress Research suggests that smokers who "use" cigarettes primarily to reduce such uncomfortable feelings as tension and anxiety ("negative affect") may be more likely to smoke under stress, and consequently may find cigarettes harder to give up (Ashton & Stepney, 1982; Biener, 1987; Russell, Peto, & Patel, 1974). Accordingly, women, who more commonly report smoking to reduce negative affect, may be at particular risk for habitual stress-related smoking.

At every age group, women appear to have more difficulty than men in quitting smoking (Stoto, 1986). Specifically, women appear less likely than men to report a wish to quit (Blake, Pechacek, Klepp, Folsom, Jacobs, & Mittelmark, 1984; Frerichs, Anashensel, Clark, & Yokopenic, 1981), to actually quit (Stoto, 1986), and to maintain abstinence after quitting (Gritz, 1980).

The reason for gender difference in quit rates is unclear. It may be due to differences in length of time smoking (Cleary, Hitchcock, Semmer, Flinchbaugh, & Pinney, 1986; Gritz, 1980) or the tendency of men to switch from cigarettes to cigars (Jarvis, 1984). Women also may be more likely to view smoking as an effective

means of weight control (Gritz, 1986), and in fact are more likely than men to cite fear of weight gain as a reason not to quit (USDHHS, 1980). However, it may also be the case that women, more then men, use cigarettes as a means of coping with feelings of distress. This "use" of smoking to manage distress may make it more difficult for women to give up cigarettes, and may make women more vulnerable to the effects of stress on smoking behavior.

In support of this notion, gender differences have been reported in the degree to which stress and distress affect or are believed to affect smoking. Women appear more likely than men to cite emotional stress as a reason for smoking (Frith, 1971; O'Connor, 1980) and are more likely to cite stressful events as precursors of relapse after quitting (USDHHS, 1980). Also, in Ikard and Tomkins's (1973) study, women were much more likely than men to describe themselves as "negative affect" smokers and to smoke more under more stressful laboratory conditions.

Studies of stress and smoking among working women provide further support for Biener's (1987) hypothesis. For example, in some professional groups women smokers now outnumber men (e.g., Biener, Abrams, Follick, &

Hitti, 1986; Sorenson & Pechacek, 1986; USDHHS, 1980). This may be due to women's greater likelihood of holding stressful jobs or perceiving their jobs as stressful. Such a hypothesis was tested by Biener and colleagues (Biener et al., 1986) in a sample of 700 male and female hospital employees. Results of this study showed that women reporting a high degree of job strain (i.e., high demand, low control) were more likely to be smokers than were women who described their jobs as low-strain. Notably, level of job strain did not predict smoking status for professional males.

Biener (1987) suggests that gender differences in negative affect- or stress-related smoking may be attributable, in part, to physiological factors, i.e., differences in the biochemical actions of nicotine which increase the chemical addictiveness of cigarettes for women. As discussed by Biener, nicotine metabolism is known to be affected by many factors, including stress (Schachter et al., 1977b). Also, women appear to excrete nicotine more rapidly than men (Beckett, Girod, & Jenner, 1971). Thus, women may, compared to men, experience sharper drops in blood nicotine levels when under stress. Furthermore, such rapid drops might be experienced as distress-producing cravings or

withdrawal. Increased smoking then would relieve the distress, thereby reinforcing smoking behavior and enhancing a dependence on nicotine. Though only speculative, such gender-specific patterns might explain, in part, why women would both report and show increased smoking under stress and distress. It also might explain why women are more likely than men to describe themselves as addicted to cigarettes (Eiser & Van Der Pligt, 1986).

Summary

This introduction has reviewed evidence for a positive relation between stress and smoking. It has been noted that both stress and smoking appear to influence the development or exacerbation of illness, and, further, appear to influence one another in a variety of ways. As smoking is known to be an important risk factor for the leading causes of death in this country, a deeper understanding of the role of stress in smoking has many implications, particularly for the successful treatment of habitual smoking.

At present, the mechanisms by which stress affects smoking are unknown. It may be that stress elicits uncomfortable emotional states that are reduced directly through smoking due to complex pharmacologic

effects of nicotine (e.g., Pomerleau & Pomerleau, 1984). However, while the psychopharmacology of nicotine clearly is instrumental (and apparently necessary), other factors appear important as well. For example, smoking may be used by smokers to regulate emotional arousal in varying conditions of stress and nonstress (e.g., Eysenck, 1973; Mangan & Golding, 1978). Alternately, stress may heighten the metabolism and excretion of nicotine in the body, resulting in greater cravings for cigarettes (e.g., Biener, 1987; Schachter et al., 1977b,c; Schachter, 1977, 1978).

A more behavioral approach suggests that stress-related smoking simply reflects habit, i.e., stress may represent a learned cue for smoking, conditioned through repeated pairings of cigarette consumption with uncomfortable or emotionally arousing situations (Ashton & Stepney, 1982). The association may be fostered or strengthened by performance-enhancing or emotion-reducing effects of nicotine which counteract a typical or learned response to stress, or actually enhance adaptation to an undesirable situation by increasing alertness and controlling fear or anxiety (Hall & Morrison, 1973; Leventhal & Cleary, 1980).

Finally, stress-related smoking may reflect individual differences that encourage smoking under states of high arousal or negative affect (e.g., Biener, 1987; Ikard & Tomkins, 1973). Such factors as gender, reported reasons for smoking, trait anxiety, accumulation of stressors, and/or social support all have been found important in smoking behavior, and may influence the relation between stress and cigarette smoking for a given individual.

Though the above hypotheses all are plausible, none has yet been found to fully explain the smoking behavior of humans exposed to stress. However, further research into the phenomenon of stress-related smoking may uncover more precise mechanisms by which stress affects smoking. As smoking occurs on a daily basis, investigation of the role of daily events and mood states appears to be a logical next step. Results of the one study that has directly investigated this issue (Conway et al., 1981) strongly suggest an association between a high level of daily stress and increased smoking. Such findings invite further research into the role of daily minor stress in smoking, and, further, factors that might influence that relation.

PURPOSE OF STUDY

One purpose of this study was to determine whether increases in self-reported daily minor stress and/or "negative affect" (as defined by scores on a state anxiety questionnaire) were associated with changes in daily smoking behavior in adult habitual smokers. Another purpose of this study was to examine whether specific variables were predictive of a positive association between daily stress or anxiety and daily cigarette consumption. The following questions were addressed:

1. What is the relation between scores on a daily minor stress inventory and number of cigarettes consumed per day? Prior research (e.g., Conway et al., 1981) suggests there is a significant positive association between amount of self-reported minor stress and number of cigarettes consumed per day.

2. What is the relation between self-reported daily mood state, as measured by the State form of the State-Trait Anxiety Inventory, and number of cigarettes consumed per day? Prior research (e.g., Biener, 1987; Conway et al., 1981; Ikard & Tomkins, 1973) suggests there is a significant positive relation between anxious mood and number of cigarettes consumed.

3. Is there an association between daily stress and mood state in their relation to daily smoking frequency? Stress is thought to produce its impact on smoking behavior by increasing arousal which is in turn reduced through increased cigarette consumption. In this study it was therefore expected that both daily stress scores and daily mood state scores would be significantly correlated with number of cigarettes consumed per day.

4. Does gender predict the relation between daily stress scores and number of cigarettes consumed? Likewise, does gender predict the relation between daily mood state scores and smoking frequency? Given prior research (e.g., Biener, 1987; Ikard, Green, & Horn, 1969), it was expected that women, compared to men, would show a stronger relation between self-reported daily stress or state anxiety scores and daily cigarette consumption.

5. Do smokers' self-reported reasons for smoking, as measured by a smoking motives questionnaire, predict the relation between daily stress and/or mood state scores and daily cigarette consumption? Prior research (e.g., Ikard & Tomkins, 1973; Joffe et al., 1981) suggests that smokers who report smoking primarily to

reduce feelings of distress produce a stronger relation between daily stress and/or anxiety scores and daily smoking frequency.

6. Does level of trait anxiety predict the relation between daily stress and/or state anxiety scores and daily smoking frequency? Prior research suggests that while smokers in general cannot be distinguished from nonsmokers on the basis of trait anxiety or neuroticism alone (Ashton & Stepney, 1982; Parkes, 1984), anxiety-prone or -reactive smokers may be more likely than are less anxious smokers to increase the number of cigarettes consumed under higher daily stress (e.g., Conway et al., 1981).

7. Do higher scores on an inventory of recent major life events predict a positive association between daily stress or anxiety scores and daily cigarette consumption? Prior research (e.g., Gottlieb & Green, 1984) has found that smokers report increased cigarette consumption during periods of greater recent life stress. Also, increased levels of job stress have been found associated with increased smoking intensity (Weiman, 1977; Westman et al., 1985). Accordingly, it was expected that high scores on a measure of recent major life events would predict a positive relation

between daily stress or state anxiety scores and number of cigarettes consumed.

8. Do scores on a social support questionnaire predict the relation between daily stress and/or anxiety scores and daily smoking frequency? Prior research suggests that higher social support is associated with less vulnerability to adverse effects of stress (Krantz et al., 1985; Vaux, 1988). Also, research suggests that smoking cessation is better maintained by those with higher social support (e.g., Horwitz et al., 1985; Mermelstein et al., 1983). A combination of low stress and high social support appears to increase the likelihood of continued abstinence after quitting (Ockene et al., 1982), while a combination of high stress and low support has been found to have the opposite effect (Caplan et al., 1975; Westman et al., 1985). Accordingly, it was expected that low social support scores would predict a positive relation between higher daily stress and/or anxiety scores and daily cigarette consumption.

METHOD

Subjects

Subjects included 55 moderate to heavy smokers over the age of 17, recruited for the study from various sites and geographic regions. Due to concerns about potential differences from "normal" habitual smokers in cases of very infrequent and very heavy smoking, recruitment was restricted to those smokers who regularly consumed between 10 and 40 cigarettes per day. However, 4 of the 55 subjects did not meet this criterion. Three subjects who had verbally estimated smoking an average of 10 to 40 cigarettes per day were found to smoke, on average, less than 10 cigarettes per day, and one subject smoked greater than 40 cigarettes per day.

Subjects were excluded from the study if they were illiterate, psychotic, presently using marijuana, presently in treatment for alcohol abuse/dependence, actively trying to quit smoking (i.e., not presently in program; not using nicotine patch), and/or unwilling to participate for the full 21 days.

As proposed, the first subjects recruited for the study were employees from two major medical centers in New York City, Bellevue Hospital and the New York

University Medical Center. Smokers at these sites were recruited in three ways: through signs posted in employee high-traffic areas, through memos distributed to hospital Department Heads, and by word of mouth (i.e., recruitment of potential subjects by current subjects and by employees who were colleagues and friends of the investigator). Using these methods, approximately 64 potential candidates for the study were identified. However, unexpectedly, the completion rate for subjects was low. Only 30 of the first 64 potential subjects actually correctly completed the study. Two could not be reached to schedule an initial screening. Eighteen refused to participate when told they would be asked to self-monitor for 3 consecutive weeks. Another 15 dropped out (for various reasons) after agreeing to participate. One subject turned in her materials but completed the study incorrectly.

Because of these problems of refusal and dropout, other methods of recruitment then were attempted. Additional subjects were recruited from other sites in New York and Louisiana, primarily through word of mouth (i.e., current subjects and friends and relatives of the investigator assisted in recruiting and served as liaisons between potential subjects and investigator).

These efforts yielded another 51 potential subjects, of whom 22 completed the study, 5 refused, and 23 dropped out. Also, one potential subject completed the study but her data was lost in transit to New York.

Instruments

Informed Consent

Two separate forms documented that subjects were duly informed of the nature, subject responsibilities and potential costs and benefits of participating in the study. (See Appendices A and B)

Sociodemographics Questionnaire

A one-page questionnaire assessing various sociodemographic characteristics was designed for the study. Items included age, gender, marital status, education, occupation, and socioeconomic level. (See Appendix C)

Medical and Smoking History

This questionnaire included general smoking and medical history items and was designed to describe the subject sample. (See Appendix D)

Reasons for Smoking Questionnaire

The Reasons for Smoking Questionnaire (RFS) (Ikard, Green, & Horn, 1969) is a 23-item self-report instrument developed from the Tomkins model of smoking

to assess various reasons for smoking. Scores are based on relative ratings for each of six smoking motives, including negative affective reduction (NAR), psychological addiction (PA), habitual smoking (HS), sensorimotor manipulation (SMM), stimulation smoking (STM), and pleasure smoking (PLS). Respondents rate each item of the RFS using a 5-point Likert-type scale ranging from 1 ("Always") to 5 ("Never"). From these ratings smoking motive scores are computed by dividing the mean item rating on each motive scale by the mean item rating for all items.

Two early studies (Ikard & Tomkins, 1973; Leventhal & Avis, 1976) provided support for the validity of the RFS, showing that the scale factors could predict smoking behavior in response to specific environmental manipulations. Factor analytic studies of the RFS and modified versions also find the six motive factors appropriate and generally consistent in composition (Bosse, Garvey, & Glynne, 1980; Coan, 1973; Costa, McCrae, & Bosse, 1980; Mausner & Platt, 1971; McKennell, 1970).

Some studies comparing self-reported with self-monitored smoking motives have shown less consistency than the factor analytic studies in the motive

categories represented on the RFS (e.g., Joffe et al., 1981; Shiffman & Prange, 1988; Tate & Stanton, 1990). However, NAR appears to be one of the more reliable motives (Joffe et al., 1981; Shiffman & Prange, 1988). In the present study, only the NAR score was used in formal data analysis. (see Appendix E)

Social Readjustment Rating Scale

The Social Readjustment Rating Scale (SRRS) (Holmes & Rahe, 1967) is a standardized questionnaire consisting of 43 different life change events. Scores reflect the total amount of (weighted) life change in the previous year. Each item on the SRRS is assigned a mean value representing the degree of life adjustment expected of or required for that event. These weights are based on the judgements of 394 subjects who rated items using an arbitrary value of 50 for one event, marriage, as an anchor.

In general, the SRRS appears to be an adequate measure of recent major life change events. It has been found to predict both medical and psychiatric illness in a number of retrospective and prospective studies (e.g., Holmes & Masuda, 1974; Rahe & Arthur, 1978). Evidence for its reliability is found in studies showing adequate test-retest stability over

various time periods ranging from 2 weeks to 9 months (Bieliauskas, 1982). Also, the perceived stressfulness of SRRS items has been found quite consistent across diverse populations and cultures (Bieliauskas, 1982). (See Appendix F)

Social Support Questionnaire

The Social Support Questionnaire (SSQ) (Sarason, Levine, Basham, & Sarason, 1983) is a widely used self-report instrument that assesses both support resources and appraisals. Most of the 27 items refer to emotional support. Questions sample a wide variety of situations in which social support might be important (e.g., "Who do you feel really appreciates you as a person?"). Respondents provide up to 9 names per item, and also rate, on a scale of 1 to 6, their degree of satisfaction with support in each situation. Scores on social network size (SSQ-N) and support satisfaction (SSQ-A) are yielded by averaging ratings across questions.

Sarason et al. (1983) report excellent internal consistency and good stability for both the social network size and support satisfaction scores of the SSQ. SSQ-N has shown a modest association with support satisfaction and with relatively low depression and

hostility. For women, higher SSQ-N scores have been associated with extraversion and with lower anxiety. Females reporting lower support satisfaction on the SSQ have been found to report higher anxiety, depression, hostility, and neuroticism. For males, a lower SSQ-N score is associated only with depression.

Subjects with higher SSQ-N scores have reported better coping with negative life events and more focus on positive events. Also, higher SSQ-N scores are associated with greater internal locus of control and self-esteem. Subjects with higher SSQ-A scores report more optimism about life and more interpersonally-focused hopes for the future.

The SSQ is limited by its almost exclusive focus on emotional support, its length, and its failure to assess network composition or quality of relationships (Vaux, 1988). However, its strengths include extensive psychometric data, use of multiple items, and attention to support satisfaction. (See Appendix G)

Daily Cigarette Tally Forms

Daily cigarette tally forms were designed for the present study. These forms were similar in format to those used in smoking cessation programs, with one form reserved for each day of the study. The form, which

was small enough to be folded into a cigarette pack, was carried by the smoker each day. It was divided into columns and rows representing all 24 hours of a day, and to complete it subjects simply made checkmarks for each cigarette smoked next to the time of day at which the cigarette was consumed. (See Appendix H)

Self-monitoring of smoking behavior has been found to provide accurate and reliable data (e.g., Joffe et al., 1981; Leventhal & Avis, 1976; Shiffman & Prange, 1988). Minimal initial reactivity effects sometimes are observed (McFall & Hammen, 1971).

State-Trait Anxiety Inventory

The State-Trait Anxiety Inventory (STAI) (Spielberger, Gorsuch, & Lushene, 1970; Spielberger, 1983) is a brief self-report measure of anxiety. The revised scale (Form Y) has psychometric qualities superior to those of earlier versions and is based on the responses of 5000 additional subjects (Spielberger, 1983).

The STAI is comprised of two separate scales which measure two conceptually different dimensions of anxiety. State anxiety (STAI-S) is viewed as a transitory emotional condition characterized by feelings of tension, apprehension, nervousness, worry,

and autonomic arousal. Trait anxiety (STAI-T) is conceptualized as a relatively stable tendency to perceive or appraise situations as stressful (i.e., as dangerous or threatening), and to respond to these situations with elevations in state anxiety (Hersen & Bellack, 1988).

Each scale of the STAI consists of 20 items. The S scale asks subjects to rate the intensity of their subjective feelings of anxiety "right now" for each item, using a 4-point scale ranging from 1 ("not at all") to 4 ("very much so"). The STAI-T asks respondents how they generally feel, rating the frequency of their experience for each item. Again, respondents use a 4-point scale ranging from 1 ("almost never") to 4 ("almost always").

Spielberger (1983) reports a strong psychometric foundation for the STAI. Studies of the STAI's factor structure show that individual items from the T and S scales load on distinctive trait and state anxiety factors. Both the S and T forms have high internal consistencies, with alpha coefficients around .90 for the normative samples. The stability of the T scale is good, with test-retest coefficients ranging from .73 to .86 from one hour to 104 days between administrations.

The test-retest reliability of the S scale is lower, as expected for a measure of fluctuating anxiety.

The T scale of the STAI has been found to correlate well with other commonly used measures of trait anxiety. Coefficients are in the range of .73 to .85 (median of .80), suggesting good concurrent validity (Hersen & Bellack, 1988). Support for the construct validity of the STAI is found in numerous studies showing changes in S scores as a function of changes in situational stress (Spielberger, 1983, 1985), and in findings of significantly higher mean scale scores for psychiatric patients as compared to normals (Spielberger, 1983).

The STAI has been used widely in both research and clinical practice across diverse populations, and has been used in over 300 treatment studies in the past decade (Hersen & Bellack, 1988). The instrument is applicable with normals, various clinical populations, elderly subjects, and children or adults with a sixth grade reading level or higher.

In the present study, both the trait (STAI-T) and state (STAI-S) scales were used. Raw scores were used in analysis of data. (See Appendices I and J)

Daily Stress Inventory

The Daily Stress Inventory (DSI) (Brantley & Jones, 1989; Brantley et al., 1987) is a 58-item self-report inventory measuring the frequency and impact of daily minor events. Respondents rate the perceived stressfulness of each item that occurred in the preceding 24 hours, using a 7-point scale. Higher ratings indicate higher stress. Three basic scores are yielded. The Event score is simply the number of items endorsed that day. The Impact score is the sum of the impact rating values from all items endorsed. The I/E Ratio, calculated by dividing the Impact score by the Event score, represents the average stress impact rating for that day. In addition to these basic scores, 5 content clusters have been identified (Brantley & Jones, 1989).

The DSI may be used to examine stress over a 1-day period or on a weekly basis. Scores from a single day are considered state measures of stress, while ratings over several days reflect more stable stress frequency and impact. Comparison data for days and weeks, in the form of percentile and t-scores, is provided for normal adults, college students, and medical patients.

Brantley and Jones (1989) report good psychometric properties of the DSI. Alpha coefficients for the Event and Impact scores are reported as .83 and .87, respectively, indicating acceptable internal consistency. Reliability coefficients for daily and weekly Event and Impact scores suggest that the frequency of minor stressful events tends to be moderately stable over short time periods but more variable from week to week, while perceived impact of events tends to vary from day to day, with more stability over longer periods. In serial administration, initial DSI scores may be slightly inflated due to a novelty or self-monitoring effect, but this does not appear to carry over into remaining days (Brantley, Cocke, Jones, & Goreczny, 1988).

Evidence for the concurrent validity of the DSI has been found in studies correlating DSI scores with global stress ratings (Brantley et al., 1987) and biochemical indices of stress (Brantley, Dietz, et al., 1988). Support for convergent and discriminant validity also are reported by Brantley et al. (1987). Evidence for the construct validity of the DSI is found in statistically significant relations between daily stress and symptom exacerbations in respiratory

disorders (Goreczny et al., 1988; Nathan et al., 1988), Crohn's disease (Garrett, Brantley, Jones, & McKnight, 1991), and sleep disturbance (Rubman, Brantley, & Jones, 1988). (See Appendix K)

Procedure

Subjects were recruited for the study using the methods described above. Where possible, subjects met individually with the investigator prior to starting the study. However, for logistical reasons, in many cases this could not be done (e.g., subjects lived outside of New York City). Those subjects who could not meet personally with the investigator communicated with her through their liaisons and/or by phone and mail.

Subjects deemed appropriate for the study were given explicit written instructions along with their packets (see Appendix L). For the sake of consistency, the same written instructions were included in all research packets, whether subjects communicated with the investigator in person or by mail. Also, with the distant subjects, the recruiting contact persons continued to serve as liaison between subject and investigator, distributing research packets when necessary and further explaining details of the study.

Research packets contained all materials needed for the study, including written instructions, informed consent forms, the initial assessment battery, and self-monitoring materials. The initial battery consisted of a sociodemographics questionnaire, a medical and smoking history questionnaire, the Reasons for Smoking scale, the Social Support Questionnaire, the Social Readjustment Rating Scale, and the Trait form of the State-Trait Anxiety Inventory (STAI-T). A second SRRS also was included in the packet, to be completed with regard only to those events occuring during the 3 weeks of self-monitoring. The daily self-monitoring materials in the packets included 21 Daily Cigarette Tally forms, 21 STAI-S questionnaires and 3 one-week DSI pamphlets. Finally, subjects were given large envelopes in which to place empty packs of cigarettes consumed during the 3 weeks of the study (as a check on accuracy of self-monitoring of cigarette intake).

After receiving their packets, subjects first completed the preliminary materials and then, on the first Monday after completing these materials, began self-monitoring their daily smoking, stress, and state

anxiety. Thus, all subjects began monitoring on a Monday and finished on a Sunday.

During the monitoring period subjects filled in their Daily Cigarette Tally forms throughout the day, evening and night. In addition, each night before going to bed subjects completed one STAI-S and one column (day) of the DSI. This procedure was followed each day for 21 consecutive days. On the last night of the 3-week self-monitoring period, subjects completed the second SRRS.

After completing the study subjects returned all materials, including (when available) empty cigarette packages, to the investigator, either in person or by mail. As compensation for participating in the study, subjects were paid $25.00 in cash after their completed materials were turned in. They also were offered feedback concerning their smoking patterns, and this was relayed to them in written form following their completion of the project.

RESULTS

Sample Characteristics

A series of univariate procedures was conducted on the sociodemographic and historical measures and the preliminary predictor variables to yield descriptive data about general subject characteristics. These results are summarized below, and are presented in some cases in Table 1.

Sociodemographics

Of the 55 subjects who completed the study, 38 (69.1%) were female and 17 (30.9%) were male. Ages ranged from 17 to 65, with a mean age of 36.15 (+/- 11.54). Racial composition of the sample was 21.2% African-American, 69.2% Caucasian, and 9.6% Hispanic.

In terms of marital status, 43.4% of subjects were married or cohabitating, 32.1% were single, and 24.5% were separated, divorced or widowed. The majority of subjects (69.8%) held professional or semiprofessional jobs; the rest were unemployed, in school, or working in unskilled labor positions. Only 3.8% of subjects had less than a high school degree. 35.2% had a high school diploma or its equivalent. 22.2% of subjects had some college, 11.1% had a college degree, and 27.8% held a graduate degree. Income level varied, with most

subjects earning between $20,000 and $30,000 per year. 22.9% of subjects earned less than $15,000 a year, and 31.3% earned over $30,000 a year. No subjects were on public assistance.

Medical Status

The majority of subjects (over 80%) reported no current or past major medical problems. Only 20.4% reported presently taking medications of any type (prescription or over-the-counter).

Alcohol Use

Most subjects (74.1%) reported drinking alcohol at least occasionally. Twelve subjects (22.2%) reported never drinking alcohol, and 2 subjects (3.7%) reported drinking alcohol on a daily basis.

Self-Reported Smoking Patterns

Subjects reported consuming an average of 21.7 (+/- 11.1) cigarettes per day. The mean number of years smoking was 19.2, +/- 11.3 years. The average number of previous attempts to quit was 1.75. Few subjects (7.5%) reported regularly smoking extra light cigarettes. Most said they smoked light (39.6%) or medium (30.2%) cigarettes. Another 22.6% of subjects reported smoking cigarettes with a heavy tar and nicotine content.

About half (49.1%) the subjects were the only smokers in their households. Thirty-six percent lived with one other smoker and 15% lived with 2 or more smokers. The majority of smokers in the sample were free or able to smoke at work and at home. Only 11.1% of subjects said they could not find a way to smoke at work, and only 5.6% were unable to smoke at home. On average smokers were awake and free to smoke 11.05 hours per day on weekdays, 14.9 hours on Saturdays, and 15.1 hours on Sundays.

Self-Assessed Influences on Smoking

On the smoking and medical history questionnaire, subjects were asked to rate, on a scale of 0 to 4, the degree of influence of various variables on their smoking. Using this scale, around 60% of subjects thought their smoking was influenced either a lot or a great deal by, respectively, daily aggravations and major life events. Thirty-nine percent thought their smoking was strongly influenced by mood. Forty-eight of 55, or 90.6% of subjects in the sample, believed they were addicted to cigarettes.

SRRS-2

Forty-seven subjects completed a second SRRS to assess major life events occurring during the actual

study. While the SRRS-2 was not used in the multiple regression procedure, univariate analysis of SRRS-2 scores revealed a mean score of 107.02, with a range of 0 to 371 and an SD of 106.6. According to Holmes and Rahe (1967), SRRS scores of less than 150 suggest low major stress; thus, scores for major life events during the study itself were, on average, within the low to moderate stress range. Over 50% of subjects scored lower than 150, and 15 subjects reported no major events at all during the study.

Individual Subjects' Correlations

Results of the correlation analyses for individual subjects' centered scores are presented in Table 2, which illustrates the large extent of individual variability in stress-smoking associations both between and within subjects and across daily stress measures. It also can be seen from this table that while a significant proportion of subjects (35%) smoked significantly more under greater daily stress, the majority did not, and a small group (4%) smoked less. This suggests that the group of subjects whose stress and smoking scores were significantly positively correlated account largely for the obtained significant

Table 1.
Summary of Descriptive Data for Preliminary Variables

Variable	N	Min	Max	Mean	SD
# YEARS SMOKING	53	3.00	54.00	19.25	11.29
# CIGS/DAY	52	8.00	70.00	21.71	11.06
# TIMES QUIT	52	0	9.00	1.75	1.98
HOURS FREE,M-F	54	2.00	19.00	11.06	4.47
HOURS FREE,SAT	53	1.00	19.00	14.85	3.86
HOURS FREE,SUN	53	3.00	19.00	15.06	3.74
STAI-T	53	20.00	69.00	37.68	10.01
NAR	54	7.00	28.00	19.78	5.02
SRRS-1	54	0	1271.00	317.91	291.46
SRRS-2	47	0	371.00	107.02	106.61
SSQ-N	50	27.00	227.00	93.52	53.29
SSQ-A	48	27.00	162.00	143.77	28.20

NOTE. HOURS FREE, M-F; SAT; SUN = hours free to smoke on Monday through Friday, Saturday and Sunday; STAI-T = raw score, State-Trait Anxiety Inventory (Trait form); NAR = raw score, Negative Affect Reduction scale of Reason for Smoking Questionnaire; SRRS-1 = sum score on Social Readjustment Rating Scale, past year; SRRS-2 = SRRS, study only; SSQ-N = Social Support Questionnaire, Network score; SSQ-A = SSQ Appraisal score

Table 2.
Individual Subjects' Within-Subject (r) Correlations between Daily Stress Measures And Cigarette Intake

Subj	STAI-S	DSI-E	DSI-I	DSI-AIR
1	-0.1904	-0.0359	-0.0994	-0.2025
2	0.3634	0.1554	0.2478	0.0943
3	-0.2618	0.4538*	0.4369*	0.0336
4	0.6298**	0.2539	0.2429	0.2456
5	-0.1624	0.5853**	0.3085	-0.0480
6	-0.5374*	-0.1240	-0.3448	-0.3902
7	0.2120	0.2183	0.3081	0.2582
8	0.5804**	.	.	.
9	-0.6041**	0.6164**	0.6638**	0.6849**
10	0.2132	.	.	.
11	0.4313*	0.0962	0.1896	0.3431
12	0.4177	0.4564*	0.4229	-0.4342*
13	0.5808**	-0.2512	0.3644	0.5757**
14	0.1129	0.2716	0.2664	0.1252
15	-0.0133	0.0404	0.0021	0.1636
16	.	0.7647**	0.7669**	0.6122**
17	-0.2994	.	.	.
18	0.1917	0.0044	-0.0724	-0.1113
21	-0.0447	-0.0318	0.0105	-0.1113
22	.	.	.	.
25	-0.1049	-0.1795	-0.2291	-0.1461
26	0.1430	-0.0231	-0.0782	-0.1783
27	-0.0699	0.1555	-0.1478	-0.2413
28	0.0725	-0.2370	-0.1041	0.1206
29	-0.1628	0.1266	0.2123	0.2143
30	0.6246**	-0.2301	0.1011	0.4531*
33	0.3827	-0.4066	-0.4399*	-0.2750
34	-0.0323	-0.1836	-0.1514	-0.0614
35	-0.2512	-0.1144	-0.1161	-0.2246
36	0.0126	0.2207	0.2745	0.1787
37	0.1321	0.6000**	0.4379*	0.1371
38	0.1636	-0.2973	-0.0558	0.2178
41	0.2948	-0.2286	-0.1637	-0.0988
42	0.4767*	0.4413*	0.6071**	0.5321*
44	.	0.8255**	0.8698**	0.4939*

NOTE. N = 55. STAI-S = state anxiety; DSI-I = stress impact; DSI-E = stress event; DSI-AIR = average stress impact. *p $\leq$ .05 ; ** p $\leq$ .01

Table 2., Continued

Subj	STAIS	DSI-E	DSI-I	DSI-AIR
45	.	0.3141	0.2880	0.1079
47	-0.3415	0.0705	0.1750	0.1176
48	0.2366	0.2037	0.1951	0.0121
50	.	0.1560	0.5720**	0.7740**
51	0.0600	0.1040	0.2990	0.2630
52	0.0080	0.6750**	0.6350**	0.1140
53	0.5490*	0.6170**	0.5220*	0.3170
54	0.0240	-0.2020	-0.1350	0.3500
55	0.3120	-0.3290	-0.2320	0.1380
56	0.2030	0.0310	0.1450	0.3810
57	-0.2030	-0.2170	-0.1800	-0.1620
58	0.0560	0.3750	0.3300	0.1040
59	0.2370	.	.	.
60	0.5630**	0.2450	0.4600*	0.4390*
61	0.0850	0.4580*	0.4750*	0.0780
68	0.0290	-0.1210	-0.3480	-0.3300
75	-0.0820	0.4320*	0.3960	0.1920
77	0.6540**	0.1020	-0.0180	-0.2170
78	-0.3195	-0.2702	-0.2935	-0.1585
82	-0.2537	-0.1662	-0.1188	-0.0614

NOTE. N = 55. STAI-S = state anxiety; DSI-I = stress impact; DSI-E = stress event; DSI-AIR = average stress impact. *p $\leq$ .05; **p $\leq$ .01

group correlations between various stress measures and smoking.

Table 2 shows further that in some cases there were conflicting results across stress daily stress measures. For instance, two subjects showed both positive and negative stress-smoking associations, and several subjects produced positive associations on only one stress measure. On the other hand, only 4 subjects had significant negative correlations between smoking and any measure of daily stress, and 2 of these 4 also had significantly positive relations between daily smoking and another daily stress measure. Moreover, no subject had significant negative relations between smoking and more than one measure of daily stress, while 13 of 19, or 68%, of subjects who showed a significant positive relation between smoking and daily stress showed this relation on more than one stress measure. Also, almost 11% of all subjects produced significant positive relations between daily smoking and 3 or more measures of daily stress. Thus, while there was substantial individual variability in stress-smoking associations, when the effect occurred, it tended to do so consistently and in the positive direction.

Within-Subjects Correlational Analyses

A cross-sectional time series procedure (CSTS) was used to determine relations between subjects' scores on daily stress and anxiety measures and daily cigarette consumption. The CSTS procedure "centers" subjects' daily scores and simultaneously obtains the average correlations between them. Centering the daily scores removes between-subjects variability by making each subject's score the difference between his own smoking on a specific day and his average number of cigarettes smoked during the 21 days of the study. This allows the CSTS to then summarize the average, or "typical," relation between subjects' daily cigarette intake and their scores on each of the daily stress variables. Barring substantial autocorrelation (i.e., interrelation of residual error terms across days), the resulting coefficient provides an estimate of the average, or "typical," subject's within-subject relation between daily stress and smoking. In other words, the coefficient describes a typical statistical relation between increases in one day's stress and increases in that day's cigarette consumption.

Using the CSTS, multiple correlation coefficients were determined for the relations between each

subject's daily cigarette consumption (DCIG) and his/her scores on the daily stress and anxiety measures (daily state anxiety scores [STAI-S], daily stress event [DSI-E], daily stress impact [DSI-I] and daily average stress impact [DSI-AIR]). A Durbin-Watson D-statistic was used to test for autocorrelation; this test revealed that first-order autocorrelation of 1-day lagged daily error terms (i.e., autocorrelation of immediately adjacent error terms) was not substantial.

Means and standard deviations for each of the daily variables are presented in Table 3. Mean correlation coefficients from the within-subject time series analysis were derived from a matrix of means for each subject, and are presented as Standardized Beta statistics in Table 4. Autocorrelation coefficients (Rho) also are presented in Table 4. Means, standard deviations and ranges of mean (R) correlations are presented in Table 5. Table 6 shows average Z-transformed correlations between daily stress measures and daily smoking.

Although within-subject correlations between daily smoking and each of the daily stress measures were significant, most of the correlations were low (Rs ranged from .116 to .181), and the significance may

Table 3.
Means, Standard Deviations, and Ranges for Raw (Uncentered) and Centered Daily Data, including Daily Cigarette Intake and Daily Stress Variables

Variable	N	Mean	SD	Min	Max
DCIG	1154	20.62	10.75	0	78
C-DCIG	1154	0	5.24	-24.48	31.52
STAI-S	1091	35.96	11.74	20	80
C-STAI-S	1091	0	8.45	-25.14	33.05
DSI-E	1091	8.14	7.16	0	38
C-DSI-E	1091	0	3.57	-10.90	17.24
DSI-I	1091	20.19	22.14	0	158
C-DSI-I	1091	0	12.38	-53.33	86.67
DSI-AIR	1002	2.35	1.03	0.72	6.46
C-DSI-AIR	1002	0	0.73	-2.20	4.03

NOTE. For each variable, upper value represents raw value, lower represents centered value. DCIG = daily cigarette intake; STAI-S = state anxiety; DSI-E = event, DSI-I = impact, DSI-AIR = average impact rating

Table 4.
Cross-Sectional Time Series:
Within-Subject Correlations of Daily Stress Measures with Daily Cigarette Intake

Stress Measure	DF	Beta	p<	Rho
STAI-S	(1, 1038)	.116	.001	.177 (ns)
DSI-I	(1, 1038)	.181	.001	.185 (ns)
DSI-E	(1, 1038)	.141	.001	.189 (ns)
DSI-AIR	(1, 1002)	.155	.001	.196 (ns)

NOTE. STAI-S = state anxiety; DSI-I = daily stress impact; DSI-E = daily stress event; DSI-AIR = daily average stress impact; Beta = standardized estimate of correlation between stress measure and smoking; Rho = autocorrelation coefficient

Table 5.
Means, Standard Deviations, and Ranges of Within-Subjects (R) Correlations between Daily Stress Measures and Number of Cigarettes Consumed

Stress Measure	Mean	SD	Lowest	Highest
STAI-S	.10	.31	-0.604	0.654
DSI-E	.13	.32	-0.407	0.826
DSI-I	.16	.32	-0.440	0.870
DSI-AIR	.11	.29	-0.434	0.774

NOTE. N = 50. Means reflect average within-subject correlations between each daily stress measure and cigarette intake. STAI-S = State-Trait Anxiety Inventory (State form); DSI-E = stress event; DSI-I= stress impact; AIR = average stress impact rating

Table 6.
Means, Standard Deviations, and Ranges of Z-Transformed Within-Subjects (R) Correlations between Daily Stress Measures and Number of Cigarettes Consumed

Stress Measure	Mean	SD	Lowest	Highest
STAI-S	.11	.34	-0.700	0.782
DSI-E	.15	.37	-0.432	1.174
DSI-I	.19	.37	-0.472	1.332
DSI-AIR	.12	.32	-0.465	1.030

NOTE. N = 50. STAI-S = State-Trait Anxiety Inventory (State form); DSI-E = stress even score; DSI-I = stress impact; AIR = average stress impact rating

been due to high power given the large number of observations for each daily variable. Results showed that, for subjects as a group, daily cigarette intake (DCIG) was related to daily stress impact (DSI-I) (Beta = .18, $p < .001$), average stress impact (DSI-AIR) (Beta = .16, $p < .001$) and stress event frequency (DSI-E) (Beta = .14, $p < .001$). The weakest correlation between smoking and a stress measure was that between daily cigarette intake and state anxiety (Beta = .12, $p < .001$).

Homogeneity of Slopes

The next step in the analyses used a homogeneity of slopes model to assess whether a single coefficient appropriately summarized the within-subjects associations between daily stress scores and smoking. Specifically, the model asked whether there were significant differences between subjects' individual time series stress-smoking relations. Results of the homogeneity of slopes model, presented in Table 7, showed that the "typical" equation did not adequately summarize all subjects' equations; therefore, between-subjects differences in within-subject stress score-smoking correlations appeared due to some source of variability other than random error. This suggested it

was appropriate to progress to the next phase of analysis, which used multiple correlation and regression procedures to explore potential predictors of relations between daily stress and smoking.

Regression Analysis

The next set of procedures addressed the question, what is the nature of the differences between subjects on stress-related smoking? This phase of the study sought to predict associations between daily stress and smoking. First, within-subject correlation coefficients from the time series analysis were transformed to Z-scores for use in the regression analysis as dependent variables. Then, correlation coefficients were determined for each of the main predictor variables and the "new" dependent variable, the Z-transformed correlations between daily stress measures and cigarette intake (see Table 8).

In the multiple regression procedures, four main independent (predictor) variables were entered simultaneously to calculate the proportion of variance accounted for by these variables on "stress-smoking" (i.e., the Z-transformed correlation between daily cigarette intake and the DSI and STAI-S scores). The main predictor variables entered into this multiple

Table 7.
Homogeneity of Slopes Model: Daily Cigarette Intake with Daily Stress Variables (Asking Whether Stress-Smoking Associations are Similar for All Subjects)

Dependent Variable	DF	F	p≤
STAI-S	(49,989)	1.86	.001
DSI-E	(49,989)	2.13	.001
DSI-I	(49,989)	2.49	.001
DSI-AIR	(49,989)	1.68	.003

NOTE. STAI-S = state anxiety; DSI-E = daily stress event; DSI-I = daily stress impact; DSI-AIR = average daily stress impact rating

Table 8.
Correlations between Main Predictor Variables and Z-Transformed Associations between Daily Stress Measures and Cigarette Intake: What Predicts Associations between Stress and Smoking?

	STAI-S	DSI-I	DSI-E	DSI-AIR
SEX	-0.084	0.189	0.075	0.266
NAR	0.156	-0.032	-0.053	0.093
STAI-T	0.162	-0.165	-0.167	-0.038
SRRS	0.214	-0.052	-0.115	0.009
SSQ-N	0.182	0.120	0.056	0.087
SSQ-A	0.031	0.241	0.218	0.209

NOTE. SEX = gender; NAR = negative-affect-reduction smoking scale of Reasons for Smoking Questionnaire (RFS); STAI-T = State-Trait Anxiety Inventory, Trait form; SRRS = Social Readjustment Rating Scale; SSQ = Social Support Questionnaire (N = Network, A = Appraisal score); daily stress measure (State-Trait Anxiety Inventory, State form (STAI-S) and Daily Stress Inventory (DSI) reflects Z-transformed correlation between each stress measure and daily cigarette intake

regression included Gender (M-F), Negative-Affect Smoking (NAR scale of the RFS), Recent Life Events (SRRS-1 score), and Trait Anxiety (STAI-T). Due to limited N, two measures of social support (SSQ-N, SSQ-A) were examined in a separate regression analysis.

Results of the multiple regression analyses showed that simultaneous regression of Gender, NAR, Trait Anxiety, and SRRS did not significantly predict relations between daily stress and smoking (see Table 9). The separate simultaneous regression of social support (SSQ-A, SSQ-N) also was not significant in predicting associations between daily stress and smoking (see Table 10).

Table 9.
Simultaneous Regression: Gender, Negative Affect Reduction, Trait Anxiety, and SRRS, Predicting Z-Transformed Associations between Daily Stress Measures and Cigarette Intake

Stress Measure	DF	R2	p
STAI-S	(4, 43)	.076	ns
DSI-I	(4, 43)	.056	ns
DSI-E	(4, 43)	.041	ns
DSI-AIR	(4, 43)	.079	ns

NOTE. STAI-S = Z-transformed correlation between state anxiety and cigarette intake; DSI-I = Z-transformed correlation between daily stress impact and cigarette intake; DSI-E = Z-transformed correlation between daily stress event and cigarette intake; DSI-AIR = Z-transformed correlation between average daily stress impact rating and cigarette intake.

Table 10.
Simultaneous Regression: Social Support (SSQ-A, SSQ-N), Predicting Z-Transformed Associations between Daily Stress Measures and Cigarette Intake

Stress Measure	DF	R2	p
STAI-S	(2, 41)	.037	ns
DSI-I	(2, 42)	.058	ns
DSI-E	(2, 42)	.044	ns
DSI-AIR	(2, 42)	.046	ns

NOTE. STAI-S = Z-transformed correlation between state anxiety and cigarette intake; DSI-I = Z-transformed correlation between daily stress impact and cigarette intake; DSI-E = Z-transformed correlation between daily stress event and cigarette intake; DSI-AIR = Z-transformed correlation between average daily stress impact rating and cigarette intake.

DISCUSSION

This study examined two questions concerning the relation between daily stress and cigarette smoking. First, is there a positive relation between daily stress or anxiety scores and daily cigarette intake? Second, do specific between-subject factors aid in predicting that association?

A one-group longitudinal research design was used to examine these questions. Subjects included 55 adult habitual smokers who self-monitored their daily cigarette intake and their daily stress and state anxiety for 21 consecutive days. These subjects also completed a number of preliminary questionnaires which were used in the prediction of relations between daily stress and smoking.

Based on prior research (e.g., Conway et al., 1981), it was expected that a substantial proportion of subjects would smoke more than their average number of cigarettes in association with high scores on measures of daily stress and state anxiety. It was further expected that five variables, gender, trait anxiety, negative affect smoking, recent life events and social support, would be predictive of an association between daily stress and anxiety scores and cigarette intake.

Results provided mixed support for the research hypotheses. To the first question, is there a significant, positive association between daily stress scores and daily cigarette intake, the answer clearly was yes. Findings from between-subjects time series correlation analyses showed a modest but significant "typical" correlation between daily stress impact scores and daily cigarette consumption across subjects; this signified that, for subjects as a group, daily smoking rate was in fact related to level of perceived daily stress. Also, results of within-subject time series analyses showed moderate to strong subject by subject correlations between daily stress scores and smoking. Specifically, a fair proportion of subjects (about 35%) showed significant positive associations between various daily stress scores and daily cigarette intake. Further, for 68% of those subjects showing a positive association between stress and smoking, the association was evident on two or more measures of daily stress. Thus, the present results provide evidence that daily stress is positively associated with cigarette smoking.

The above findings are consistent with prior research showing that smokers increase their cigarette

intake under various forms of stress, both in the laboratory (e.g., Mangan & Golding, 1978; Schachter et al., 1977b) and in the natural environment (e.g., Comer & Creighton, 1978; Conway et al., 1981; Schachter et al., 1978). Such results appear generalizable to the general population of habitual smokers, as the smoking behaviors and histories reported by the present subjects were quite similar to those reported by adult habitual smokers in other studies of cigarette smoking (e.g., Joffe et al., 1981; Shiffman & Prange, 1988). In addition, the present findings fit well with reports from other longitudinal studies of daily stress (e.g., Brantley et al., 1988; Caspi, Bolger, & Eckenrode, 1987; Garrett et al., 1991), which tend to show weak but significant group effects and considerable intersubject variability in specific effects of daily stress on various criterion behaviors.

Interestingly, in this study, associations between state anxiety scores and daily cigarette intake were lowest of all stress measures. For individual subjects this was sometimes the case even when other measures of daily stress were positively correlated with daily smoking. Such findings might be construed to suggest that stress but not anxiety is related to smoking, and

therefore anxiety is not the mechanism by which stress is linked with smoking. However, prior research would argue against this interpretation. Evidence from both within and outside the laboratory suggests that cigarette smoking often is elicited or increased by anxiety-arousing situations (e.g., Ikard & Tomkins, 1973; Schachter et al., 1977b; Stepney, 1980). Models of smoking relapse incorporate this premise, given the consistent observation that relapse most commonly occurs when a former smoker is in a negative mood state and/or has faced some kind of immediate stressor causing him or her distress (Borland, 1990; Brandon et al., 1990; Cummings et al., 1985; O'Connell & Martin, 1987; Shiffman, 1982). Together such findings provide considerable support for the notion that smoking is in some way positively related to anxiety. Accordingly, at this point it would be premature to assume that smoking behavior in this study was completely unrelated to anxious mood.

An alternate explanation is that in this study anxiety was in fact aroused by subjectively stressful daily events, but for some reason anxiety was not reported by subjects. Failure to report daily anxiety could be due, in part, to inconsistencies in the

measurement of daily anxiety and cigarette smoking in this study; as state anxiety was measured only once per day, at night, an anxious mood state associated with daily stressors may simply have subsided by the time the state anxiety measure was completed. It also is possible that smokers did not consistently report anxious mood in association with daily stress precisely because they smoke. In other words, smokers may not have perceived themselves as particularly anxious because their usual coping response to stress (i.e., smoking) effectively served to alleviate anxiety. This idea would be consistent with research showing that cigarette smoking is associated with both self-reported and behavioral indications of anxiety-reduction as well as inducement of feelings of calm and pleasure in smokers (Ague, 1973; Gilbert, 1979). It has been argued that smokers' reduced distress feelings following a stressor are due to nicotine itself rather than the mere act of smoking (Stepney, 1980), and this effect may be dose-dependent (e.g., Nesbitt, 1973; Stepney, 1980). Thus, research on mood-altering effects of nicotine suggests that smokers who smoke when made anxious by a stressor may find themselves in a state of nicotine-induced muscular relaxation and

perceived emotional calm; that state may be experienced by the smoker as overall tension-reduction, even if other indications of continued emotional arousal are present. The smoker thus may feel he is relaxed by smoking, whether or not he actually is. In this way, a state-anxious smoker might not show a relation between daily anxiety and smoking rate precisely because he perceives his smoking behavior as anxiolytic.

At the same time, the actual or perceived anxiolytic effects of cigarettes need not affect a smoker's retrospective appraisal of an event as having been stressful when it occurred; rather, if stress and smoking are positively associated, smoking should better correlate with the subjective appraisal of stressors' impact than with a measure of daily mood. Indeed, this is what the present findings showed; in this study, the DSI Impact score, a measure of perceived stressfulness of daily events, was the daily stress measure most strongly correlated with daily smoking. Because the Impact score represents the immediate subjective appraisal of stress, this score, rather than a daily anxiety score, might be the best measure of arousal associated with daily stressful events. For this reason, stress impact would be more

likely than state anxiety to be found significantly correlated with daily cigarette intake. This is particularly true if stressors elicit mood states other than anxiety (e.g., anger, sadness, distress).

To the second research question, what factors might predict a positive association between daily stress and smoking, the answer is more equivocal. In the present study, positive correlations between daily stress and smoking could not be explained on the basis of any of the proposed predictor variables, including gender, trait anxiety, recent major stress, self-reported negative-affect-reduction smoking and social support. Indeed, none of the coefficients between stress-smoking associations and these major predictor variables even approached statistical significance.

The failure to predict "stress-smoking" in this study is in some ways surprising, given the bulk of prior research and theory linking the proposed predictors with stress-related smoking. Take, for instance, trait anxiety, conceived by Speilberger (1983) as chronically high state anxiety. One might expect that a smoker described as chronically anxious would be hyperresponsive to stress and therefore likely to show a significant relation between daily stress and

smoking. However, in the present study, this was not the case; here, scores on a measure of trait anxiety were in no way predictive of an association between daily stress scores and smoking. The reasons for this are unclear. However, the inconsistency may involve issues of measurement similar to those discussed with regard to state anxiety. Also, as was discussed in regard to state anxiety, the anxiolytic properties of cigarettes may have further obscured any real relation between daily stress and state or trait anxiety.

It is more difficult to explain why self-reported negative-affect-reduction (NAR) as a motive for smoking did not predict correlations between daily stress and smoking in the natural environment. Given prior research in this area, it was expected that NAR scores, which reflect a smoker's belief that his smoking functions to reduce tension or anxiety, would help identify smokers who, in their daily lives, increase their smoking under stress. However, in this study, this was not the case. On the contrary, NAR scores were not at all predictive of "stress-smoking." However, a closer examination of research on the NAR scale in particular and the Reasons for Smoking Questionnaire (RFS) in general may shed some light on

this puzzle. While several studies have provided evidence for the predictive validity of the NAR scale in identifying smokers who smoke primarily to reduce tension or anxiety (e.g., Ikard & Tomkins, 1973; Joffe et al., 1981; Shiffman & Prange, 1988), findings are inconsistent. For example, while Shiffman and Prange (1988) found NAR to be one of two RFS motives to actually predict self-monitored smoking behavior, Tate and Stanton (1990) found that NAR scores were not significantly correlated with light smokers' self-monitored smoking behaviors. Across studies of this type, corresondence between NAR scores and self-monitored smoking motives typically is only moderate, ranging from .19 to .56 (Shiffman & Prange, 1988). Thus, there is, at best, only moderate correspondence between self-reported and self-monitored NAR smoking, and the fact that a smoker believes he smokes to reduce tension does not guarantee that he in fact does so. Indeed, this problem extends to all six motives assessed by the RFS. As noted by Shiffman and Prange (1988), none of the various RFS motive scales has consistently been found to correlate well with self-monitored smoking behavior. For this reason, Shiffman and Prange conclude that, in general, "the

correspondence of smoking typology measures to smoking behavior patterns is at best weak" (p. 204).

The issue of self-reported versus self-monitored smoking behavior is complicated further by most studies' use of very brief smoking self-monitoring periods as well as their use of varying instruments to measure NAR smoking. Also, across studies of self-reported and in vivo smoking motives, there are dramatic differences in the constitution of subject samples, with some studies using only adult habitual smokers (e.g., Shiffman & Prange, 1988) and others using college-age light smokers (e.g., Tate & Stanton, 1990). Accordingly, the noted inconsistency between self-reported and self-monitored smoking motives in the present study could be due to multiple factors, including weaknesses of the RFS Questionnaire and limitations of smokers to self-assess their actual smoking behavior.

That gender did not in this study predict an association between daily stress scores and smoking is surprising. However, it should be noted that in the present study female subjects far outnumbered males (38 to 17); given this discrepancy, there may have been too little statistical power to detect sex differences if

they in fact existed. However, there may be other reasons for the results. While it has been suggested that women are more likely than men to smoke to relieve tension or distress (e.g., Biener, 1987; Frith, 1971; O'Connor, 1981), such a conclusion is based largely on self-report. For instance, women are more likely than men to say they use cigarettes in response to negative affect (Frith, 1971; O'Connor, 1980; Zuckerman, Ball, & Black, 1990), to cite stress as a reason for relapse (Frith, 1971; USDHHS, 1980), and to report experiencing negative emotions when they "slip-up" (i.e., lapse or relapse) after quitting (Borland, 1990). Such gender-different reports may be construed to suggest that women smoke more under stress than do men; however, it is equally plausible that they reflect differences in self-assessment rather than actual behavior. It is known, for example, that women are more likely than men to report emotional symptoms, to seek psychiatric and medical help (Verbrugge, 1985), and to receive psychotropic medication (Biener, 1987). Perhaps this reflects womens' social conditioning to describe themselves as more emotional and more emotion-bound than men (Biener, 1987). For this reason women may be more likely to report and to perceive their smoking as

strongly influenced by negative affective states, even if the influence in actuality is only slight. Thus, women may see themselves as strongly influenced by their emotional states and therefore may believe they smoke to reduce tension, even though their self-report does not necessarily correspond to their in vivo smoking behavior. The opposite may be true for men, who may see their smoking behavior as relatively unaffected by emotional state when it in fact is. As in the case of many problem behaviors, perception of behavior may be quite different from actual behavior.

In fact, in experimental studies of stress and smoking, women do not seem to differ much from men. In a series of studies by Dembroski (1986), for example, women showed the same interactive effect of stress with smoking as did men (Dembroski, 1986). Specifically, in these studies a proportion of both male and female subjects, termed by Dembroski the "hot" reactors, demonstrated the same kind of heightened reactivity to a stressor plus smoking; in these cases it was found that cardiovascular response to a stressor or smoking was notably high, and response to the two together was significantly greater than that elicited by either factor alone. The proportion of males and females

found by Dembroski to be "hot" reactors was not substantially different. Given such findings, one might propose that a smoker's gender is actually less important than the degree to which he or she is a "hot" reactor to stress and to smoking.

It should also be noted that few studies to date have explored gender differences in real-life stress-related smoking. The most often-cited study of this type was conducted years ago by Ikard and Tomkins (1973), who found that a significantly greater proportion of female subjects as compared to males smoked during an upsetting film but not during a funny film. This finding was and still is taken as evidence that women are more likely than men to be negative-affect smokers; however, the sample size in Ikard and Tomkins' study was quite small, and similar findings have not consistently been reported in other studies. Indeed, most studies reporting gender differences in stress-related smoking are correlational (e.g., Biener et al., 1986), and as such have not firmly established that women in fact smoke more under stress than do men. Thus far, then, evidence for real gender differences and their possible underpinnings is limited. While Biener (1987) has proposed biological and sociocultural

factors that might account for gender differences in stress-related smoking, further research clearly is needed in this area.

It is perhaps less surprising that scores on a measure of social support did not predict stress-smoking associations in the present study. Social support clearly is important in relations between stress and smoking cessation (Caplan et al., 1975). However, social support does not appear to buffer against more immediate (i.e., same-day) effects of daily stress on mood (Caspi et al., 1987), and so might not be expected to predict associations between daily stress and smoking.

A similar case may be made with regard to major life events, which in this study did not predict stress-smoking associations. Major life events are believed important in the relation between stress and smoking cessation and have been linked in some studies (e.g., Gunn, 1983) to smoking relapse. However, the effects of major life events on daily mood and behavior are variable. The time series study by Caspi et al. (1987), for instance, illuminates this issue. In that study, contrary to the investigators' expectation, major life events in the last year were found to be

inversely related to a significant relation between daily stress and daily mood. The authors of that study explain this finding in the context of Fechner's Law of psychophysics; i.e., the greater subjective weight of major life events may require a much greater difference in the frequency or intensity of minor events in order for a real difference to be detected and reacted to. In other words, having experienced many and/or more intense stressful life events in the past year may decrease rather than increase one's responsiveness to minor daily events. If this is the case, high scores on a measure of major life events might not predict an association between daily stress and smoking, because high scores would render an individual less rather than more responsive to daily stressors. Alternately, major events may exert their effects independent of minor events (e.g., Garrett et al., 1991), and so may simply be unrelated to daily stress.

To integrate, it now has been argued that each of the predictor variable proposed in the present study had weaknesses that might account, in part, for its failure to predict associations between daily stress and smoking. However, in spite of this the fact remains that a number of subjects in this study did

smoke more in relation to daily stress. The next logical question, then, is, why? Of course, one only can speculate on this matter. However, various other potential predictors of "stress-related smoking" may be proposed, and this could prove fruitful in considering future research and clinical implications.

In exploring possible predictors of "stress-related smoking," attention can be turned to two main areas: between-subjects factors and within-subjects factors. The predictor variables hypothesized in the present study represent between-subject factors; these factors are presumed to somehow systematically influence relations between daily stress and smoking. For reasons yet unknown, the hypothesized factors did not predict "stress-related smoking" in this study; however, other between-subject factors might be proposed and explored empirically. For instance, personality factors conceivably could influence the degree to which a smoker responds to stress with increased smoking. Such personality factors as the Type A Behavior Pattern (TABP) (Friedman & Rosenman, 1974) have been found influential in the relation between stress and illness (Feuerstein et al., 1986), and may play some role in the association between daily

stress and smoking. There is some evidence to support this idea. Caplan et al. (1975), for example, found that smokers as compared to ex-smokers reported significantly higher scores on a measure of TABP (a chronic tendency to seek out and experience high stress) and also reported significantly more job stress. Such results suggest that leading a chronically stressful life may make it more difficult for smokers to quit, presumably because of the association between stress and smoking. Since both TABP and smoking are known risk factors for coronary heart disease (CHD), a relation between them is of particular concern. Indeed, there is some evidence that strong physiological responses to stress may enhance physiological reactivity to smoking (Dembroski, 1986), and together this heightened reactivity may place "stress-smokers" at particular risk. If "hot" reactors to psychological stress (i.e., Type A personalities) tend also to be "hot" reactors to smoking, as Dembroski's results suggest, these individuals may be more likely to show a stress-smoking association, and, most notably, may be at greatest risk for some type of smoking-related illness (e.g., CHD).

Another potential mediating factor in the relation between stress and smoking is addiction to cigarettes. Tomkins (1966; 1968) claimed that heavy smokers, who presumably are more addicted to cigarettes, should want to smoke regardless of the smoking situation, while lighter smokers should show more variation in smoking rate (Tomkins, 1966; 1968). If this is so, the less addicted smoker might be expected to show a stronger relation between stress and smoking because his smoking rate is freer to vary under differing conditions. There is some support for this idea. Frith (1971), for example, found differences between light and heavy smokers in self-reported desire to smoke under various conditions of arousal. This investigator found that heavy smokers reported a desire to smoke in both high- and low-arousal situations, while lighter smokers could be dichotomized into two groups, those who wanted to smoke more in states of high arousal, and those who reported a desire to smoke under low arousal. However, it should be noted that Frith's study involved simply asking subjects what they imagined they would do; there was no empirical test of subjects' smoking behavior under the various conditions. Given the previously discussed problems of consistency between self-reported

and actual smoking behavior, Frith's findings cannot be taken as evidence that smokers in fact behave as they imagine they might. Also, other findings on stress-related smoking and level of addiction are mixed. For instance, Caplan et al. (1975) found no difference between light and heavy smokers on measures of job stress or TABP, and Schachter et al. (1977b) found that heavy but not light smokers smoked more intensely (50% more puffs) under a high-stress laboratory condition as compared to a low-stress condition.

Of course, it also is possible that such between-subjects factors as personality style, addiction, or any of the predictor variables proposed in the present study simply cannot account for the phenomenon of "stress-related smoking." Perhaps smokers represent too heterogeneous a group. If this is the case, another way to explore relations between daily stress and smoking behavior is to examine within-subject factors, i.e., factors specific to individuals rather than to smokers as a group. These factors are not expected to be consistent across smokers and therefore are not thought likely to predict smokers' average or "typical" smoking behavior; if known, they only would predict the smoking behavior of that individual.

The importance of individual differences in smoking behavior is well-documented. It is known, for example, that the arousing and sedating effects of nicotine may vary both within and across individuals. As stated by Gilbert (1979), "the effects of nicotine on CNS activity are multiple and depend on a variety of parameters... (The) differential effects of smoking and nicotine [vary] with different doses and behavioral predispositions" (p. 646). The importance of individual differences also is found with regard to the effects of daily stress. For example, Caspi et al. (1987) used a longitudinal time series design to examine relations between daily stress and mood and possible moderators of that relation. They concluded that "the effects of daily events are not uniform; under certain conditions they are distinctly negative; under others they are actually positive... (and) the underlying process that governs the relation between any two variables is not uniform across individuals" (pp.193-194). They state further that "by observing different patterns of covariation within individuals across time, we may begin to identify the individual attributes and environmental circumstances that are most critical in the stress process" (p. 194).

Given the apparent importance of individual differences in both daily stress and smoking behavior, a strength of the present study is its illumination of highly variable subject by subject correlations between daily stress and smoking. As noted, all subjects in this study did not smoke more under stress. Rather, the significant group effect of stress on smoking was accounted for by the one-third or so of subjects who did smoke more under stress. What makes these subjects different from the others remains unknown. However, the observation invites more detailed study of those particular individuals whose smoking behavior was indeed influenced by daily stress.

Finally, comment should be made about the potential role of alcohol in daily stress and smoking, as over 75% of the subjects in the present study admitted to at least occasional alcohol use. There is a well-documented association between alcohol use and cigarette consumption (e.g., Griffiths, Bigelow, & Liebson, 1976; Istvan & Matarrazo, 1984; Zimmerman et al., 1990), and the strength of that relation may be greater with higher doses of alcohol. For example, Griffiths et al. (1976), in a laboratory study, found that smokers smoked more cigarettes on days on which

they were given alcohol than on days on which they were given placebo. Furthermore, the relation between cigarette and alcohol use in that study was dose-dependent; the higher the dose of alcohol, the more cigarettes were consumed. Such findings suggest that drinking alcohol may set the occasion for smoking. Though not addressed in the present study, an important next question is, where and how does stress fit into this picture? Do stress and alcohol act together to further prime a smoker to smoke, since each factor alone has been shown to contribute to increased smoking? This issue appears a logical next step for the future study of stress and smoking.

Given the limitations on current knowledge, it must be concluded that smoking behavior may best be understood as multiply-determined. Such between-subjects factors as gender, trait anxiety, recent life events, belief that one smokes more under stress, lower degree of addiction, etc., may to some extent encourage increased smoking under stress; however, a relation between stress and smoking also may be modified by such within-subject factors as alcohol use, and/or such contextual factors as availability of cigarettes, restrictedness of smoking environment, presence or

absence of other smokers, presence or absence of smoking cues, illness, and so on. It thus is believed that between- and within-subjects factors as well as situation-specific factors all may prove to influence whether and why a given smoker consumes a cigarette at any given time. Unfortunately, at this point, our knowledge simply is too limited to say more than this.

Future studies could shed light on roles played by various factors involved in the relation between stress and smoking. The first needed step, obviously, is replication of the present findings with a larger sample of both male and female smokers, to more firmly establish the existence of a proportion of smokers for whom increased daily cigarette consumption is positively associated with high scores on measures of daily stress. To better address the issue of state anxiety and smoking, such a study might also include assessment of more immediate emotional and situational antecedents of smoking behavior (i.e., ratings of both mood state and subjectively appraised stress prior to and following each cigarette consumed; description of setting in which cigarette was smoked). In addition, given the well-documented association between alcohol and cigarette use (Istvan & Matarazzo, 1984), it would

seem most important for future studies, where possible, to obtain detailed reports of both daily cigarette and alcohol consumption over time (i.e., specific amount of cigarettes and alcohol consumed and time period, setting and social context in which such consumption occurs). Finally, to further address the issue of prediction of "stress-related smoking," future studies might include multiple measures of "negative affect reduction smoking," as well as measures of relevant personality factors and level of both physiological and psychological addiction to cigarettes.

From a clinical standpoint, findings of this study suggest that for some smokers stress management may be particularly indicated. Because of the potentially multiplicative effects of stress and smoking, those smokers who show a strong relation between daily stress and smoking might be at particular risk for various forms of heart disease (Epstein & Jennings, 1986). This effect might be further compounded for those smokers who are notably physiologically reactive to both stress and smoking (Demobroski, 1986). Thus, in terms of disease prevention, it might prove fruitful to learn to identify "stress-smokers" for evaluation of this and other known risk factors for major illness.

The present findings also have potential implications for relapse prevention. Given the common finding that stressful situations often precipitate smoking lapse and relapse (e.g., O'Connell & Martin, 1987), one might expect such situations to be particularly high-risk for those smokers for whom stress is shown to be important in daily smoking. In light of the dismally high relapse rate among all smokers attempting cessation by formal programs (e.g., Hunt & Matarazzo, 1982), it would appear potentially quite valuable to identify "stress-smokers" when they first present for treatment so as to better provide them with appropriate relapse-prevention skills.

SUMMARY AND CONCLUSIONS

In conclusion, the present study provided renewed support for the idea that stress and smoking are related. It was found that a significant proportion of subjects did, as expected, smoke more than their usual number of cigarettes on days with greater-than- usual stress impact. Such findings lend further support to a conception of smoking as a maladaptive coping response to daily stressful events (Wills & Shiffman, 1985). In addition, the study's design further extended previous findings by looking at actual smoking behavior, in the smoker's natural environment, over a considerable period of time. This allowed closer examination of what smokers actually do in the face of real-life demands. Most importantly, the study's design permitted exploration of smokers both as a group and as individuals, and in doing so set the stage for future study of potential individual differences among smokers.

As yet there are no firm explanations as to why one smoker smokes more under stress and one does not. However, it seems increasingly clear that smokers are a heterogeneous group within which subgroups of "stress-responders" may be found. It is hoped that future

research will further describe various subgroups of cigarette smokers and will someday illuminate the mechanisms underlying associations between stress and smoking.

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APPENDIX A: LSU INFORMED CONSENT

I, _____, agree to participate in this research project. I understand that this project is being conducted as part of a doctoral degree requirement through the Louisiana State University department of psychology, in Baton Rouge, Louisiana.

I am aware of the responsibilities of research participants. I have been informed of any potential risks to me. I understand that my participation is voluntary and that I may cease my participation at any time if I so choose. I have been informed that information given by me will remain confidential, and that I will be identified for the purpose of the study only by a research number.

_____ _____

Name Date

APPENDIX B: BELLEVUE INFORMED CONSENT

NEW YORK UNIVERSITY MEDICAL CENTER
A private university in the public service
550 FIRST AVENUE, NEW YORK, N.Y. 10016
CABLE ADDRESS: NYUMEDIC
BELLEVUE HOSPITAL

INFORMED CONSENT TO PARTICIPATE IN RESEARCH

You are being asked to volunteer to be a subject in a research study. This form is designed to provide you with information about this study which you should know and to answer any of your questions.

PROJECT DIRECTOR: Fred Covan, Ph.D., and Kathy Nathan, M.A.

TITLE OF RESEARCH STUDY: Factors Involved In Smoking

☐ This research study includes procedures that may change the treatment you would otherwise receive. We hope the knowledge gained will be of benefit to you.

☒ This research study includes procedures which may not give you immediate benefits. It is hoped the knowledge gained will be of benefit to others in the future.

☐ This research study is planned to select your treatment by chance. It is not known if the treatment you will receive will be of benefit to you.

THE PURPOSE OF THE RESEARCH IS:

To better understand some of the reasons people smoke

DONATION OF BLOOD: _____ cc. (equivalent to _____ ounces).
Frequency of withdrawal: _____ . The potential risks of donating blood, none of which are likely to occur, may include pain, bruising, fainting or a small infection at the puncture site.

THE FOLLOWING PROCEDURES WILL BE INVOLVED: (IF LIMITED TO DONATION OF BLOOD, LEAVE BLANK.)

Participants will be asked to meet with a researcher to talk about the study. Participants will fill out some questionnaires about their smoking, recent events in their lives, their social support system, their mood, and their present life situation. Afterthis participants will keep records of their smoking, their mood, and their daily life events, for three weeks. Participants will also be asked to save their empty cigarette packages during this time. At the end of the three weeks participants will fill out another questionnaire about life events.

Participants will be compensated for their time with $25.00 and will also be offered feedback about their own smoking patterns.

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CONSENT TO PARTICIPATE IN RESEARCH (CONTINUED)

THE POTENTIAL RISKS OR DISCOMFORTS TO YOU ARE: (IF LIMITED TO DONATION OF BLOOD, LEAVE BLANK)

The study involves about one hour to start and then about 10 minutes per day. This may be inconvenient at times.

THE POTENTIAL BENEFITS TO YOU OR OTHERS ARE:

Benefits to you include learning more about your own smoking patterns, and financial compensation of $25.00 for completing the project.

GENERAL CONDITIONS: Should you consent to participate in this research, your identity will be kept confidential. You may change your mind at any time. Refusal to participate will not harm your relationship with the faculty and attending staff.

AGREEMENT TO PARTICIPATE

I have read the above description of the research study and general conditions (or it was read to me by: _____).

Anything I did not understand was explained to me by: _____ , and any questions I had were answered by: _____ . I certify that I am / am not (circle one) participating in another research project at this time, and have discussed the implications of such activity with the project director(s). In consideration of this understanding, I voluntarily agree to participate in this research at: ☐ NYUMC ☐ Bellevue Hospital ☐ Goldwater Hospital ☐ Other _____

Name of Subject _____ Age (If under 18) _____

WHEN THE SUBJECT IS AN ADULT

Signature of Participant or Legal Representative	Date	Print Name of Legal Representative	
Signature of Investigator	Date	Signature of Witness	Date

WHEN THE SUBJECT IS A CHILD

☐ I have solicited the assent of the child. ☐ I have not solicited assent for the following reason(s): _____

Signature of Investigator (Cathy Lathan), M.A.

☐ I agree with the manner in which assent was solicited and given by my child and I agree to have my child participate in the study.

☐ Although my child did not or could not give his/her assent I agree to have my child participate in the study.

Signature of Parent(s)	Date	Print Name of Legal Representative	
Signature of Child	Date	Signature of Witness	Date

For children between the ages of 12 and 17, their signature is generally required in addition to that of the parent or legal representative.

APPENDIX C: SOCIODEMOGRAPHICS QUESTIONNAIRE

S # _____

Name: _____

Phone #: (_____)_____

Address: _____

_____

1. Age: _____ 2. Sex: M F

3. Race: African-American White/Caucasian

Hispanic Other (_____)

4. Marital status: Married Single Separated

Divorced Widowed

Cohabitating

Other(_____)

5. Occupation: _____

6. Education: _____

(highest grade completed)

7. Approximate yearly income: _____

8. Do you receive public assistance? Yes No

APPENDIX D: MEDICAL AND SMOKING HISTORY

Name: _____ S # _____

Date: _____

Hospital/Clinic: _____

Medical History

1. Do you have any major medical problems? Yes No

   If yes, please describe:

2. Do you take any medications on a regular basis?

   Yes No

   If yes, please list:

3. In the past, have you had any of the following:

Heart disease	_____	Lung disease	_____
Cancer	_____	Liver disease	_____
Kidney problems	_____	Alcoholism	_____
Drug Abuse	_____	Other:	_____

4. Are you currently having any other physical symptoms?

   Yes No

   If yes, explain:

5. Do you ever drink alcohol? Yes No

   If yes, how often and how much?

Smoking History

6. How long have you been smoking? _____ years

7. How many cigarettes do you smoke each day? _____

8. What brand do you usually smoke? _____

9. What is the tar-nicotine content of this brand? (circle one)

   Extra Light Light Medium Heavy

10. Have you ever tried to quit smoking? Yes No

    If yes, how many times? _____

    How long did you stop? _____ (longest ever)

    How did you stop? (e.g., program, nicotine gum, on your own) _____

    Why did you resume smoking?

    _____

    _____

11. Are you considering quitting now? Yes No

    If yes, why?

12. Are you presently deliberately cutting down?

    Yes No

13. Has your doctor told you to quit smoking? Yes No

    If yes, why?

14. Are you currently having any physical problems directly related to smoking (for example, coughing, shortness of breath, stained teeth)? Yes No

If yes, what are they?

15. Are you presently in treatment for alcoholism? Y N

16. Do you regularly use marijuana? Y N

17. Do you regularly use any other nonprescription drug?

Y N

Smoking Habits and Preferences

18. Does your work environment prohibit smoking? Yes No

    If yes, can you get around this? Yes No

    Please explain:

19. Are you prohibited from smoking at home? Yes No

    If yes, can you get around this? Yes No

    Please explain:

20. Does anyone else in your household smoke? Yes No

    If yes, who?

21. Approximately HOW MANY HOURS each day are you actually free to smoke (that is, you are not working in a smoke-free environment, not sleeping, etc.)?

    _____ hours per day, Monday - Friday

    _____ hours per day, Saturday

    _____ hours per day, Sunday

22. Using the scale below, rate the degree to which each of the following influences your smoking:

0----------1----------2----------3----------4

not at all a little a fair amount a lot a great deal

	Degree of Influence
The weather	0 1 2 3 4
Time of day	0 1 2 3 4
Setting (e.g., at home)	0 1 2 3 4
Alcohol	0 1 2 3 4
Coffee	0 1 2 3 4
Food	0 1 2 3 4
Daily aggravations	0 1 2 3 4
Major life changes or events	0 1 2 3 4
Physical health (being ill)	0 1 2 3 4
Mood	0 1 2 3 4
Sex	0 1 2 3 4
Exercise	0 1 2 3 4
Money	0 1 2 3 4
Other: _____	0 1 2 3 4

23. When are you more likely to smoke?

(circle all that apply)

When you first awaken in the morning

When driving

Before eating

After eating

At work

With coffee

With alcohol

While on the phone

When watching TV

At a party

At a bar

When reading

When trying to diet

When in a bad mood

When something bad happens

When with other smokers

Late at night

When angry

When bored

Other: _____

24. Do you think you are addicted to cigarettes?

Yes No

APPENDIX H: DAILY CIGARETTE TALLY

Morning		Afternoon/Evening		Night	
TIME	#CIGS SMOKED	TIME	#CIGS SMOKED	TIME	#CIGS
5:00	_____	1:00	_____	9:00	_____
6:00	_____	2:00	_____	10:00	_____
7:00	_____	3:00	_____	11:00	_____
8:00	_____	4:00	_____	12:00	_____
9:00	_____	5:00	_____	1:00	_____
10:00	_____	6:00	_____	2:00	_____
11:00	_____	7:00	_____	3:00	_____
12:00	_____	8:00	_____	4:00	_____

NAME: _____ DATE: _____

M T W TH F SAT SUN

Did you drink alcohol today?
If yes, when (approximate hours) and how much?

Comments:

Note. Size of Tally enlarged for this paper; Daily Cigarette Tally used in study could be folded three-ways to fit into a regular-sized cigarette package.

APPENDIX L: INSTRUCTIONS TO RESEARCH SUBJECTS

This study examines factors involved in smoking over time. To be included one should smoke between one-half pack and two packs a day, and should not be trying to cut down or change his or her smoking in any way during the study period. Also, persons who regularly use marijuana or alcohol and persons with a major medical condition that affects their smoking (example: asthma) are not eligible. All subjects completing the study will be given $25.00 as well as an individual assessment of their smoking patterns. Data will be kept strictly confidential.

To begin the study, sign the NYU-Bellevue Consent Form and the LSU Consent Form, in the front of the first packet in your folder. Then complete, at your leisure, the other questionnaires in that packet. Make sure to check for questions on both sides of each sheet, as the two questionnaires at the end both are front-back. Please try to answer all questions, unless doing so would make you uncomfortable in some way. Also, do not worry about adding up your own scores on any of the questionnaires.

On the first Monday after getting your folder, you will begin recording the cigarettes you smoke. To do this, take one of the small Daily Tally sheets (there are three sets in your folder), fold it three-ways, and place it, with a small pencil, in your cigarette pack as a reminder to record each cigarette. Then each time that you smoke, place a checkmark in the appropriate time slot on the tally sheet. If you finish a pack of cigarettes, put the Daily Tally sheet in the new pack, and save the empty pack until the end of the study (so daily tallies can be verified). Thus, you should have one tally sheet for each day of the study, as well as all your empty cigarette packs, by the end of the three weeks. If you give away cigarettes please note this on the tally sheet where it says "Comments." Also, if you drink alcohol on that day please note this, as well.

On the same day that you begin recording cigarettes smoked, you will begin recording daily events and mood. At night before going to bed please complete one Self-Evaluation Questionnaire (there are three sets of seven in the folder), and fill out one column of the Daily Stress Inventory (DSI) (there are three in the folder, one for each week). Write the date at the top, and then, for each item that happened that day, rate how stressful it was for you, using the scale provided. At the end of the three weeks you should thus have completed 21 Self-Evaluation Questionnaires and all three DSIs. Again, do not worry about adding up your scores.

Finally, on the last day of the three weeks, complete the second Social Readjustment Rating Scale in your folder, noting how many times each event listed happened to you WHILE YOU WERE DOING THE STUDY. Do not worry about adding up scores.

At the end of the three weeks you will be paid $25.00. Keep all your completed questionnaires etc., in the folder, and put your empty cigarette packages in a plastic or paper bag.

VITA

Kathryn Leigh Nathan was born in New Orleans, Louisiana. She graduated cum laude from Isidore Newman School in 1981. In May of 1985 she graduated with distinction from the University of Virginia, where she majored in Psychology and minored in English. She then began a doctoral program in clinical psychology at Louisiana State University.

In May of 1988 Ms. Nathan was awarded a Master of Arts degree in Psychology from Louisiana State University. Shortly thereafter, she presented a version of her Master's thesis, entitled "Daily Stress and Disease Severity in Asthma," at the 1988 Annual Meeting of the American Psychological Association, in Atlanta, Georgia. This paper received the 1988 Graduate Student Research Award in Health Psychology by Division 38 of the American Psychological Association.

Ms. Nathan completed her internship in clinical psychology at NYU-Bellevue Hospital Center, in New York City. She presently resides in Manhattan, where she is completing a one-year post-doctoral fellowship in clinical psychology at St. Vincent's Hospital.

DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Kathryn Leigh Nathan

Major Field: Psychology

Title of Dissertation: Daily Stress and Smoking

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

February 5, 1993